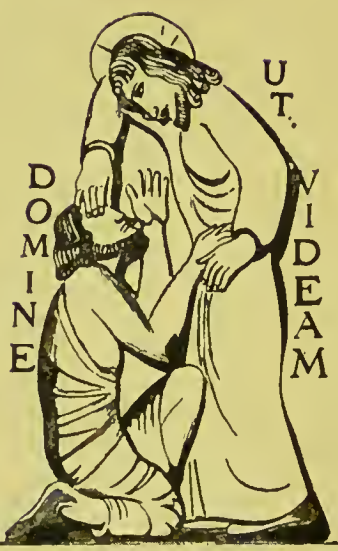
THE EYE

AS AN AID IN

GENERAL DIAGNOSIS.

LINNELL.



THE EYE

AS AN AID IN

GENERAL DIAGNOSIS.

A

HAND-BOOK

FOR THE USE OF

Students and General Practitioners.

BY

E. H. LINNELL, M. D.

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Dedicated to

J. E. LINNELL, M. D.,

my FATHER, my PHYSICIAN,

and my FRIEND.

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INTRODUCTION.

EXAMINATION of the eyes affords valuable aid not only in the diagnosis of diseases of the central nervous system, but also of constitutional affections and diseases of other organs.

It has long seemed to the writer that this subject was too much neglected by the general practitioner. The record of the pulse, temperature and respiration, urinary analysis, etc., are among the every day routine methods of diagnosis, but the indications furnished by the eye are too little understood, and too often overlooked.

When this treatise was commenced, it was with the conviction that such a work was demanded, as there was then no similar treatise in the English language. Much of it was completed before the appearance of the encyclopaedic work of Knies entitled "Relations of Diseases of the Eye to General Diseases," and I have freely availed myself of any information contained therein, which had not previously come to my knowledge.

It has not been my purpose to enumerate all the eye symptoms which may be associated with the various constitutional and local diseases, but rather to emphasize such as are of direct importance in the way of diagnosis, and to present such data so as to be of ready reference and practical value.

The book has been written from the standpoint of the specialist for the student and general practitioner. It embodies the personal experience of the writer during a general practice of twenty years, and fifteen years experience in the treatment of ocular diseases, in addition to extensive reading.

I have not endeavored to go into the symptomatology or differential diagnosis of the various ocular affections which are discussed, further than the purpose of the work demanded, or into the treatment of such affections. To do otherwise, would be to write a treatise upon Ophthalmology. The book is simply what its title indicates, namely, a Handbook of Diagnosis, and as such I hope it may find a place among the reference volumes of the student and the busy family physician who aims to keep abreast of the times.

E. H. LINNELL, M. D.

Norwich, Connecticut, April, 1897.

PART FIRST.

THE EYE SYMPTOMS OF NERVOUS AND CONSTITUTIONAL DISEASES.

CHAPTER I.

AFFECTIONS OF THE EYELIDS, CONJUNCTIVA, ORBIT, EYE-BALLS, SCLERA, AND CORNEA.

EYE LIDS.

In examining the lids for indications of general disease, it is important to notice the color of the skin, the presence or absence of thickening or oedema, of inflammation of the ciliary margins, of neoplasms, the movements of the lids, the existence of dilatation or contraction of the palpebral fissure.

More or less anaesthesia of the skin of the lids, with false localization of sensation is a symptom of locomotor ataxia.

A pigmentation of the skin of the lids accompanies Addison's disease of the supra-renal capsules. In other cases it is symptomatic of uterine or hepatic disease. It is sometimes associated with abdominal growths. Blue rings around the eyes may accompany menstruation in debilitated individuals. When they disappear with the cessation of the menstrual flow, the symptom is of no special importance, and does not indicate organic disease.

A swollen oedematous non-inflammatory condition is indicative of nephritis and should lead one to examine

the urine, even in the absence of anaemia, debility and other concomitants of renal affections. It is also present in general hydraemia and in heart disease. It accompanies suppuration within the orbit, but in the latter condition, it is sharply circumscribed by the bony edge, and this circumstance will at once distinguish this form of oedema, from that accompanying the affections previously mentioned, in which it is not distinctly circumscribed, but gradually merges into the healthy skin of the eye-brow or the cheek.

Oedema of the lids is also suggestive of trichinosis. It frequently accompanies that disease, and may be one of its very early manifestations.

Thickening and swelling of the lids may be an initial manifestation of myxœdema.

Eczema of the skin of the eyelids, especially of the ciliary border, is frequently dependent upon a scrofulous diathesis, but in many cases it is caused by eye strain, due to refractive errors, and is cured by the prescription of suitable glasses. When neither of the above causes exists, the presence of an obstinate eczema of the margins of the lids should lead to an examination of the urine, for this is a not infrequent accompaniment of diabetes.

Styes, it is well-known, are frequently associated with disorders of digestion, and with menstrual irregularities, but they are often, also, the result of eye strain, and are cured by correcting errors of refraction. I have

a patient who had a succession of styes for many months. She was not conscious of any imperfection of her vision, but a weak cylindrical glass before each eye cured the styes, and also a headache from which she had frequently suffered.

A tubercular nodule sometimes develops in the tissue of the eyelid, simulating a large inflamed chalazion (a retention cyst of the Meibomian gland). A knowledge of this fact may be of advantage in treatment.

It should also be remembered that the initial lesion of leprosy may develop in the lid in the form of nodules, which are hard and insensitive, of a whitish or pale yellow or reddish color, accompanied with more or less infiltration of the sub-cutaneous tissue. Anaesthetic, whitish patches may also appear.

In studying the muscular conditions of the lids, both paralytic and spasmodic conditions are of diagnostic importance. I will consider these conditions separately as affecting the orbicularis and the levator of the upper lid.

General debility, especially in old age, frequently produces a laxity of the skin of the eyelids, with a deficient innervation of the orbicularis, causing, secondarily, eversion of the eyelids, and epiphora from malposition of the puncta, and a chronic conjunctivitis. Hence such conditions suggest a lowered vitality and a need for constitutional treatment, and careful diet to promote nutrition.

A true paralysis of the orbicularis produces the condition known as lagophthalmus, in which the patient is unable to close the eyes. It is associated with paralysis of the muscles of the face, owing to their common innervation by the facial nerve, and indicates the peripheral nature of such a paralysis, for in 90 per cent of facial paralyses of central origin the orbicularis and frontalis muscles escape. An explanation of such exemption is found in the assumption that the fibres of the nerve which supply these muscles arise from a separate nucleus from that of the rest of the nerve. The possibility of ear disease and of syphilis should be borne in mind. A paresis of the orbicularis, causing imperfect closure of the lids, sometimes occurs in posterior spinal sclerosis—locomotor ataxia—and should awaken suspicion of this disease. Sometimes, in very ill persons, a paralysis of the eyelids is simulated by a lack of sensibility of the cornea and conjunctiva, so that the natural stimulus to close the eyes is lost.

Diminished frequency of winking from the same cause also occurs in Basedow's disease, or exophthalmic goitre, and is known as Stallwag's sign, or Dalrymple's symptom.

A spasmodic action of the levator of the upper lid (Abadie's sign) is another of the symptoms of Basedow's disease, and Von Graefe first called attention to another characteristic feature of the affection, viz.: a spasm of Mueller's muscle. This consists of a

few unstriped muscular fibres in the cellular tissue of the orbit, innervated by the sympathetic. Its contraction causes a widening of the palpebral fissure, and interferes with the associated movements of the eyeball and the lid. This is noticed in looking downward, when the lid lags behind, so that a white stripe of exposed sclera is seen between the edge of the cornea and the lid. This is a characteristic symptom of exophthalmic goitre, and, together with Stelwag's sign, gives the peculiar staring expression to such patients, and renders the exophthalmus more noticeable. Starkey found Von Graefe's sign present in all but 12 of 613 cases of exophthalmic goitre. It is an early symptom, and may, for a considerable time, be the only feature of the case. It may affect only one eye, or may be more marked on one side. In testing for this sign it is best to have the patient lying upon his back. His gaze should be directed at some object, first held directly above his face, then slowly moved downward toward his chest, when the deficient movement of the upper lid becomes apparent.

The opposite condition, that of narrowing of the palpebral fissure, was described by Jackson as a symptom of posterior spinal sclerosis, and is caused also by an opposite lesion, viz. : a paralysis instead of an irritation of the sympathetic. It is sometimes designated a "sympathetic ptosis."

A tremulous action of the lids accompanies paralysis agitans.

A spasm of the orbicularis is, in the majority of cases, an expression of photophobia. The lids are closed to shut out the light which is painful, owing to inflammation or hyperaesthesia. When no inflammation exists, a cause of the hyperaesthesia and its resultant spasm may sometimes be found in eye strain from refractive or muscular anomalies, especially the former. It may also result as a reflex neurosis dependent upon a a sexual or intestinal irritation.

Spasmodic winking, or nictitation of the lids, is also a nervous manifestation, which likewise may result from errors of refraction, may accompany or precede general chorea or, by reflex action, may be associated with disorders of the nose, teeth, or digestive organs.

We distinguish various forms of ptosis or inability to raise the lids. There is a congenital form, usually associated with diminished power of raising the globe, and supposed to be due to a "congenital, central defect." In other cases there is a lack of development of the levator muscle. The writer is acquainted with a Swedish family where all the children exhibit this peculiarity. In such cases there is an overaction of the frontalis muscle which gives the individual a peculiar anxious expression. There is a form designated as "morning ptosis," which occurs after sleep in debilitated individuals. The levator is relaxed during sleep, and in these cases a few minutes are requisite to enable the person to recover the voluntary contraction of the muscle.

There is, also, an hysterical ptosis. The latter may be unilateral or bilateral, and is associated with spasm of the orbicularis, which latter is particularly marked when the patient is told to look upward. A transient ptosis has been known to accompany an irritation of the fifth nerve, from the extraction of a tooth.

These various forms of ptosis are to be distinguished from true paralysis of the levator muscle. The latter may result either from a peripheral neuritis, such as occasionally occurs after exposure to cold, and from alcoholism, or from a lesion of the third nerve anywhere in its course from its nucleus of origin to the orbit. An isolated paralysis of the third nerve is usually of nuclear origin, but it may be caused also by a circumscribed lesion of the cortex of the frontal lobe of the cerebrum, just in front of the fissure of Rolando. The fibres of the third nerve decussate in their intracerebral course, so that destruction of this centre causes ptosis of the opposite side. The cortical centres for the motor nerves of the eyeball are at a considerable distance from this point, and hence it is not strange that ptosis sometimes occurs as an isolated paralysis, and such an occurrence is a valuable point in the location of cerebral symptoms having this association.

Ptosis is sometimes an accompaniment of paralysis agitans and not infrequently is present in tabes, so that its association with these affections needs no other explanation.

The significance of ptosis when occurring with loss of function of the other motor nerves of the eyeball, does not need separate discussion here. It is hardly necessary to mention the simulated ptosis due to cicatrices caused by erysipelas, or to cellulitis orbitae, periostitis, and adhesions between the lid and eyeball. Such a condition could hardly deceive the most careless and superficial observer.

CONJUNCTIVAL AFFECTIONS.

In considering affections of the conjunctiva as affording suggestive hints in general diagnosis, both that of the lids and of the eyeball is understood.

It is desirable to note especially the presence of inflammation, swelling general and circumscribed, and of new growths.

The various forms of conjunctivitis, both palpebral and ocular, are frequently idiopathic, from exposure to cold, dust, strong light, etc., but very often they are an indication of a scrofulous diathesis, or of indigestion, and many cases only yield to treatment after careful prescription of glasses. Conjunctivitis also is frequently dependent upon nasal catarrh. Hence, when it does not readily yield to suitable treatment, it will often be of advantage to carefully examine the nose for the exciting cause.

The phlyctenular form, characterized by the formation of small vesicles and pustules, is very frequently

caused by nasal disease. These phlyctaenae develop very frequently along the margin of the cornea, or sometimes on the cornea itself. Knies says nearly 90 per cent. of such cases in children are from such a cause.

It is hardly necessary for me to mention the obvious causal relations of severe purulent conjunctivitis, and the poison of gonorrhoea, or other infectious secretions. I have known an eye to be lost from panophthalmitis resulting from infection with pus from an ulcerated tooth.

Conjunctival catarrh, as is well-known, accompanies the prodromal stage of measles, and it is a frequent manifestation in the early stage of epidemic cerebrospinal meningitis.

It is important to recognize the diphtheritic and croupous forms of conjunctivitis. In both we have the development of a pseudo-membrane, and the same distinction is to be made here, as in the throat, in the differential diagnosis of the two affections. In the latter the membrane is superficial and can be removed, leaving a raw, bleeding surface, while in the former it is interstitial, infiltrating the whole thickness of the mucous-membrane which is pale and bloodless, owing to obstruction of the circulation by the pressure of the exudate. A microscopic examination would be decisive. In diphtheria of the conjunctiva, the constitutional symptoms are often severe, and the swelling and induration of the whole lid is much more extensive and firmer than in conjunctival croup.

Spontaneous conjunctival hemorrhages are indicative of an atheromatous condition of the arteries, and, especially in elderly people, should lead us to appreciate the danger of cerebral hemorrhage, and to adopt precautionary measures.

Such hemorrhages are not infrequent in diabetes, owing to the vascular degeneration which occurs in that disease. Hemorrhage into the conjunctiva has been observed in persons suffering with cholera, and is considered of serious import.

Oedema of the conjunctiva.—A uniform swelling of various degrees of tension, with or without inflammation, frequently accompanies meningitis. The exudation may reach the orbit through the optic foramen, or it may produce a venous stasis and subsequent exudation by pressure upon the ophthalmic vein. It is an important indication of exudation in the cranial cavity. It is frequently an early manifestation in both the epidemic cerebro-spinal form, and in purulent basilar meningitis.

When there is an abrasion of the conjunctiva it may become inoculated with the bacilli of tuberculosis. This may occur through infection from the nose, skin (lupus is now classed as a tuberculous affection), or lungs; or the initial affection may be thus caused in a healthy individual exposed to infection. It then becomes important to recognize it, so that prompt destruction of the nodule may prevent constitutional infection. An ulcer develops at the point of inoculation, with a

hard base often covered with granulations. Yellowish-red nodules develop in the vicinity, giving a granular appearance somewhat similar to trachoma, within which the microscope reveals the tubercle bacilli. Later the lymphatic glands of the face and neck become involved.

According to Eklund, leprosy may also originate in the conjunctiva through the use of towels, etc. In view of the increasing prevalence of that disease in this country, a knowledge of the fact may possibly be of service. It is said to be caused by a micro-organism similar in appearance to that of tuberculosis. The primary nodules, pale, yellowish or reddish, insensitive and hard, increase in size and invade the other structures of the eye.

ORBIT.

A periostitis or caries of the orbit, when not traumatic, should awaken suspicion of either tuberculosis or syphilis, as it is usually caused by one or the other. Michael says that "many cases of spontaneous cellulitis orbitae in children and young people may be attributed to a tuberculous infection of the cellular tissue."

EYEBALL.

The general expression of the eye will rarely escape notice. The bright, lustrous, staring eye in febrile conditions and in mental excitement is familiar to all. So, also, is the dull, expressionless stare of mental hebe-

tude or actual dementia, and, less markedly, of typhoid conditions, *et cetera*.

A protrusion of the eyeball, exophthalmus, is one of the cardinal symptoms of Basedow's disease. It is usually bilateral, but not invariably, or it may be of unequal extent in the two eyes. It may be due to orbital growths, to suppuration within the orbit, or to aneurism of an orbital artery or of the internal carotid. In the latter case the pulsation of the aneurism will be communicated to the eye, and be perceptible to the examiner's fingers. Sometimes also a *bruit* can be detected by the aid of the stethoscope. Pressure on the cavernous sinus or the ophthalmic vein may cause sufficient venous stasis to produce protrusion of the eye, in which case the eye can be readily replaced by gentle pressure. An exophthalmus developed in a patient of the writer after using atropin for the examination of re-refraction, and subsided under application of gentle pressure. Exophthalmus in the new-born is sometimes caused by retro-ocular hemorrhage. Such a case came under my observation. The child was seen the day after its birth, and presented a marked proptosis, which disappeared in a few days under gentle pressure, and treatment prescribed for a co-existent conjunctivitis.

Every tissue of the eye at times affords points of diagnostic importance. After a brief consideration of the sclera and cornea I will discuss disorders of motility at some length, inasmuch as spasmodic and paralytic

affections of the ocular muscles are of extreme importance in cerebral localization.

The icteroid coloration of the sclera is too well recognized to need comment, but when occurring independently of hepatic affections, it is suggestive of Addison's disease of the supra-renal capsules. Idiopathic scleritis is a rare affection. Rheumatism and syphilis are the most frequent causes of inflammation of the sclera and of the episcleral tissue. Syphilitic gummata may develop primarily in the sclera, but they usually invade this tissue from the uvea. A knowledge of the cause of such affections will suggest a suitable line of treatment. Tuberculous nodules also occasionally occur in the sclera. In doubtful cases the sclerotic may at times afford conclusive evidence of death by the existence of a desiccated patch within the palpebral fissure, either at the inner or outer side and below the cornea.

CORNEA.

The cornea reveals a constitutional dyscrasia very readily. The occurrence of phlyctaenulae at the edges of the cornea in scrofulous children has been already alluded to in speaking of conjunctivitis. Ulcers and abscesses of the cornea are also frequently seen in strumous subjects.

A parenchymatous inflammation of the cornea, characterized by a diffuse infiltration into the deeper layers of the membrane, giving an appearance of ground

glass, which might be mistaken for a cataract by a careless or inexperienced observer, is very characteristic of inherited syphilis. It is rare in the acquired form of the disease. It most frequently appears between the sixth and fourteenth years. Mauthner estimates that $\frac{4}{5}$ of the cases of interstitial keratitis are syphilitic. It may accompany infectious diseases, and it has been ascribed to rheumatism. Where corneal affections develop in children who are free from scrofulous or syphilitic taint, the teeth should be examined, as there seems sometimes to be a relation of cause and effect between dental caries and such affections. Tuberculous nodules may develop primarily in the marginal zone of the cornea, and later in the cornea itself. Ulceration of the cornea is not of infrequent occurrence in diabetes, and is an indication of debility, and it should be remembered that slight injuries in elderly and feeble individuals, which readily heal in persons in good health, are very apt to break down into necrotic suppurative processes, and hence demand especial care.

In anaesthesia of the trigeminus, accompanying facial paralysis, or in conditions of profound depression, as in cholera, typhoid, etc., a condition known as neuroparalytic keratitis develops. An ulceration occurs which spreads rapidly and leads to destruction of the membrane and irreparable blindness. This is due to injury to the cornea, in consequence of its lack of sensibility. Because of this lack of sensibility, particles of dust, etc.,

are not felt, and the cornea is not protected and kept moist by the natural frequent closure of the lids. The occurrence of such cases gave rise to the theory that there existed special trophic fibres in the trigeminus which presided over the nutrition of the cornea, but this was disproved when Snellen demonstrated that simple protection of the eye was all that was necessary to prevent such traumatic and infectious ulcerations. This condition is somewhat analagous, but is to be differentiated from, the excessive dryness and loss of transparency of the cornea known as xerosis or desiccative keratitis, which occurs after excessive loss of fluids, as in cholera, childbed, etc.

Anaesthesia of the cornea occurs in locomotor ataxia, so that in some instances the membrane may be touched without producing winking of the eyelids. Sometimes also there is a false localization of sensation, so that a touch upon the cornea is referred to the external or internal canthus.

A malarial cachexia may manifest itself by an inflammation of the cornea. Noyes says, in an editorial note in Knies' "The Eye in General Diseases:"

"Keratitis, as the result of malaria, is not infrequent, and presents features which are more or less typical. It attacks, by preference, the epithelium and superficial layers, is non-suppurative; ulcerations are superficial. It is chronic in duration. There is often anaesthesia of the surface. The opacity is apt to run

in streaks, yet may present itself in patches. One will find marked tenderness of the supra-orbital nerves as they pass out of the orbit, which is the most valuable pathognomonic sign, and when occurring with the conditions described, indicates the absolute necessity of quinine in effective doses as an adjunct to local treatment."

Leprous nodules occasionally develop in the corneal tissues.

An opacity and insensibility of the cornea is an evidence of death which might be of service in a doubtful case.

CHAPTER II.

AFFECTIONS OF THE EXTERNAL OCULAR MUSCLES.

Paralytic and spasmodic affections of the external eye muscles occur with various intra-cranial and spinal diseases, and while they are usually associated with other symptoms which indicate with more or less precision the situation and nature of the lesion, the eye symptoms alone will often furnish valuable diagnostic indications.

A spasm of a given muscle, or group of muscles, is produced by an irritation of a locality whose destruction causes a paralysis of the same part. It is self-evident that a nerve may be excited or depressed in any portion of its course from its ultimate distribution to its termination in the cells of the cerebral cortex. Hence an accurate knowledge of the minute anatomy of the ocular motor nerves is a necessary preliminary to a discussion of this branch of the subject. Therefore for the sake of clearness, I will repeat here what may be familiar.

The third, fourth, sixth, facial and sympathetic, it will be remembered, are the motor nerves of the eye. With the exception of the sympathetic, they can all be traced to the gray matter of the aqueduct of Sylvius,

and to that of the fourth ventricle. The nuclei of origin of both third nerves anastomose freely, and that of the abducens communicates with the third and the adjacent nuclei of the seventh and ninth. From here the fibres of the third, or motor-oculi-communis, pass through the crus cerebri and emerge at its inner side just in front of the pons. Those of the fourth wind around the crus and emerge at its outer edge, and those of the sixth pass downward through the pyramids of the medulla and appear at the lower posterior edge of the pons. Thence these three nerve tracks extend along the base of the brain, pass through the cavernous sinus, where the third and sixth receive communicating filaments from the carotid plexus, and enter the orbit through the supra-orbital fissure. The terminal fibres of the third nerve are distributed to the superior, inferior and internal recti muscles, the inferior oblique, the levator palpebrae superioris, the ciliary muscle, and the sphincter iridis. The individual bundles of fibres innervating these various muscles have been traced to a series of centres in the aqueduct of Sylvius, which are arranged in the following order from before backward, viz. :

1. Ciliary muscle.
2. Sphincter iridis.
3. Levator palpebrae.
4. Rectus internus and superior.
5. Rectus inferior and obliquus inferior.

These several nuclei of origin may be simultaneously or individually diseased, as will be mentioned later in detail.

The fourth nerve, or patheticus, is distributed to the superior oblique, and the fibres of the sixth pass to the external rectus.

The facial nerve arises from the floor of the fourth ventricle, and it emerges at the posterior border of the pons, passes through the Fallopian canal of the petrous bone, and makes its exit through the styloid foramen, from which point it spreads out over the face, and, among other muscles, is distributed to the frontalis and the orbicularis palpebrarum.

The motor fibres of the sympathetic arise from the lowermost portion of the cervical cord, and, in their course to the eye, pass through the superior cervical ganglion and the carotid plexus, and are distributed to the unstriped fibres in the orbit constituting Müller's muscle, whose contraction dilates the palpebral fissure, and to the dilator fibres of the iris.

From these contiguous nuclei of origin fibres pass to the higher centres in the cerebral cortex, governing the voluntary and associated movements of the eyes. Bearing thus in mind the origin and course of the fibres of the different motor nerves of the eyeball, a study of the variety and relations of the ocular motor disorders enables one to arrive at a more accurate diagnosis of the situation of a lesion than would be possible without such knowledge.

It is important to remember, in making a diagnosis of a cerebral affection from ocular symptoms, that

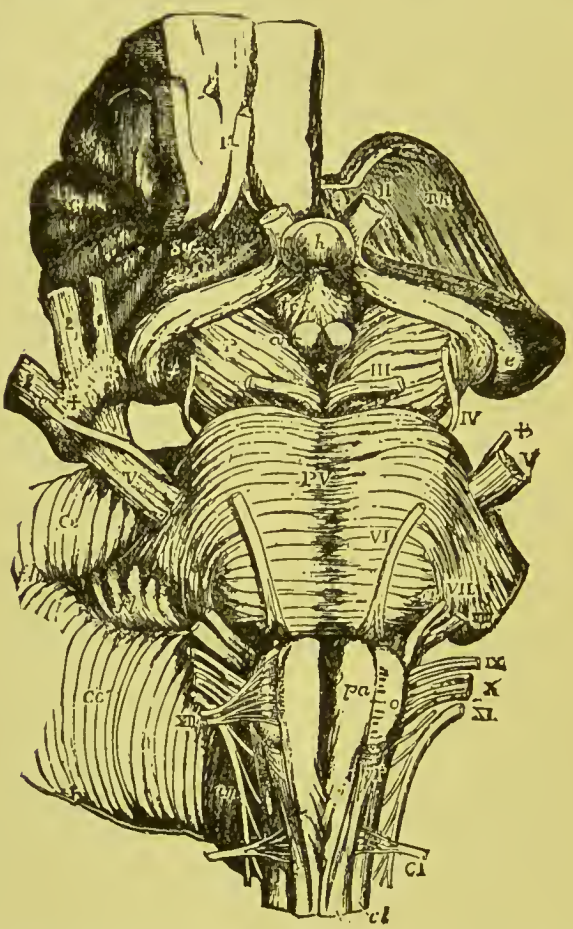


FIG. 1.—Diagram of Base of the Brain and Cranial Nerves, Pons and Medulla taken from "Diseases of the Eye," by H. D. Noyes, M. D. I to XII, The Cranial Nerves; *Th.* optic thalamus; *h.* pituitary body; *tc.* tuber-cinereum; *a.* corpora albicantia (mammillaria); *P.* pes pedunculi; *i.* internal geniculate body; *e.* external geniculate body; *PV.* pons Varolii; *pa.* anterior pyramid of medulla; *o.* olive; *d.* decussation of anterior pyramids; *ca.* anterior column of spinal cord; *cl.* lateral column of spinal cord; *Ce.* cerebellum; *fl.* flocculus of cerebellum; × locus perforatus posticus; + (on the left side), ganglion of Gasser; + (on the right side), motor root of trigeminus.

parts remote from the primary lesion frequently suffer temporarily. This is especially true of apoplectic and

inflammatory conditions. The permanent symptoms, therefore, are the only ones upon which an accurate localization may be based.

PARALYTIC AFFECTIONS.

I will first consider the significance of paralytic affections of the extrinsic eye muscles, leaving for future discussion paralysis of accommodation and of the iris. The diagnosis of a total paralysis of an ocular muscle presents no difficulty. It is at once evident, by the inability to turn the eye in the direction of the paralyzed muscle, and by the squint caused by the unopposed action of its opponent. It is otherwise when there is incomplete loss of function. Often there is no apparent loss of motion, and the paresis is only manifested by the resulting diplopia. That is only noticed when the eye is turned in the direction of the weak muscle and results from inharmonious action with its fellow of the opposite eye, so that there is inaccurate binocular fixation of an object. In consequence, the retinal images are not formed upon corresponding portions of each retina, and the aerial projection of the images are not identical, and so an apparent doubling of the object results. The following directions for detecting a paresis of the ocular muscles are so admirably expressed that I cannot do better than to quote them, although I regret that I cannot state the name of the author who formulated them:

"In determining which rectus muscle is affected, there are two things to be determined. First, the part of the field in which the doubling occurs; secondly, the eye which sees the furthest removed object. The affected muscle is always on that side of its eye towards which the doubling takes place, and it belongs to the eye that sees the furthest removed object. To illustrate: Suppose that the patient has single vision immediately in front and to the right, but, on moving the candle in the horizontal plane to the left, doubling takes place. At once we are able to say that the affected muscle is either the external rectus of the left, or the internal rectus of the right eye. Since the affected muscle belongs to the eye that sees the object furthest removed, it only remains for us to cover one eye or the other, and learn from the patient which object disappears. On covering the right eye, if the object furthest removed is blotted out, then the affected muscle is the internal rectus of the right; if the contrary, then the paretic muscle is the external rectus of the left.

"If the doubling takes place in the vertical plane, single vision being below and immediately in front, but doubling taking place as the candle is raised above the horizontal plane, the affected muscle is the superior rectus of one eye or the other. It belongs to the eye that sees the higher object. If, on covering the right eye, the higher object disappears, then it is

the superior of the right; if the contrary, it is the superior rectus of the left.

"Paralysis and paresis of an oblique muscle requires a more careful study, and yet it is comparatively easy to locate the affected muscle. If a candle be held vertically before such a pair of eyes there would be doubling, one image being vertical and the other leaning, the vertical candle being seen by the non-affected eye, while the leaning candle is seen by the eye to which is attached the affected muscle. If the left eye sees the leaning candle, and the inclination is towards the right, the affected muscle is the superior oblique of that eye. If it leans toward the left, the affected muscle is the inferior oblique of that eye. The vertical meridian of the eye affected is always turned in the opposite direction to the leaning of the candle."

It is desirable to make a distinction between paralysis of one or several eye muscles and ophthalmoplegia. By the latter term is understood the simultaneous paralysis of those muscles of both eyes which are concerned in conjugate movements, for instance, the external rectus of the right and the internal rectus of the left eye, by the aid of which both eyes are simultaneously directed upon an object on the right side. Such conjugate paralyses are of necessity of intra-cerebral or cortical origin, and a distinction should be drawn between associate and conjugate paralyses, as will be referred to later.

The existence of conjugate contraction of the eyes is also of diagnostic significance. This is reserved for future discussion in connection with spasmodic affections.

In a given case of external ocular paralysis, our first endeavor is to ascertain whether it is peripheral, that is located within the orbit, basilar, that is in the path of the nerve along the base of the skull, or intra-cerebral (fascicular, nuclear or cortical).

I. PERIPHERAL PARALYSES.

An ocular paralysis due to a disease of the orbit such as tumor, periostitis, cellulitis, etc., will be unilateral and associated with other symptoms such as pain, inflammation of lids and conjunctiva, exophthalmus, etc., and there will be an absence of evidence of intra-cranial and cerebral or spinal disease, such as implication of other nerves, vertigo, drowsiness, pupillary phenomena, headache or mental aberration. In cases of traumatic origin, the evidence of injury, or the history of the case will be decisive. Sometimes the association of the muscles affected will settle the diagnosis of its peripheral nature, as for instance, if the inferior oblique and the internal ocular muscles (iris and ciliary) are affected without implication of the other external muscles. This is evident if we remember that the iris and ciliary muscles are supplied by the short ciliary nerves, coming from the ciliary ganglion situated in the back of the

orbit, between the optic nerve and the external rectus, and that the motor fibres of the ciliary ganglion are derived from the branch of the motor-oculi which supplies the inferior oblique, after its division from the main trunk. Diphtheria is a cause of ocular paralysis which is often due to a peripheral neuritis.

EXTERNAL OCULAR PARALYSES OF BASILAR ORIGIN.

A lesion at the base of the brain producing ocular paralysis would almost of necessity affect other nerves—the fifth, seventh, optic and olfactory, either simultaneously or successively—so that the diagnosis would rest upon the association of other symptoms, especially of hemianopic visual disturbances from compression of one optic tract. The absence of such symptoms would afford negative evidence against the basilar situation of the causative lesion. When the trigeminus is paralyzed by a basilar affection, it is preceded by neuralgia, which is not the case when it is of cortical origin. An isolated paralysis of the third nerve due to an intra-cranial basilar lesion is possible, but it affords no data for a positive diagnosis. The absence of implication of the iris and ciliary muscle would counter-indicate a basilar origin, as all the fibres from the separate nuclei of origin are united when the nerve emerges from the crus.

Simultaneous paralysis of both third nerves might result from the pressure of a tumor, a syphilitic

gumma for instance, between the cerebral peduncles (crura cerebri), or from an aneurism of the posterior cerebral artery. A thrombosis of one cavernous sinus, or an aneurism of one internal carotid might cause total paralysis of the muscles of one eye, but as the ophthalmic division of the fifth nerve also passes through the cavernous sinus, anaesthesia of the parts supplied by this nerve, lids, conjunctiva, nasal fossae and integument of the eyebrows, forehead and nose, would accompany the motor disorder.

INTRA-CEREBRAL PARALYSES, INCLUDING AFFECTIONS OF THE CRURA (FASCICULAR PARALYSIS), OF THE PONS, NUCLEI AND CORTEX. ALTERNATING PARALYSES.

The fibres of the motor nerves of the extremities decussate in the medulla, those originating in the right hemisphere being distributed to the left side of the body and vice versa. The motor nerves to the ocular muscles decussate higher up. Hence the existence of "alternate paralysis," by which is meant paralysis of one or both eyes on one side, and of the extremities on the other side, indicates that the lesion is between the medulla and the point of decussation of the ocular motor nerves, viz.: either in the crus or the pons.

The ocular symptoms may be limited to one eye or affect both. A lesion (hemorrhage, abscess, tumor), limited to one crus or to the base of the brain imme-

diately beneath it is extremely rare. It would occasion paralysis of the motor-oculi on the side opposite the hemiplegia. If the iris and ciliary muscle were not implicated, the lesion could be definitely located within the crus, because the fibres from the anterior nucleus are, in that location, still considerably separated from the others.

A paralysis of one external rectus and of the extremities on the opposite side indicates a lesion at the posterior edge of the pons. So, also, an isolated lesion of one sixth nerve, must be referred to a lesion in one side of the pons. Since the sixth and facial have a common origin in the gray matter in the floor of the fourth ventricle, a nuclear lesion must affect both nerves.

An alternating paralysis caused by a lateral lesion of the pons is manifested by ophthalmoplegia, or loss of the conjugate movement of both eyes toward the sound side of the body. The eyes would deviate toward the paralyzed side from unrestrained action of the opposite muscles, and therefore the lesion is on the opposite side of the pons from that toward which the eyes deviate. Conjugate deviation of the eyes also occurs as a temporary symptom in most cases of apoplexy, but when the hemorrhage is in one of the cerebral hemispheres, the paralysis is not alternate, because the lesion is above the decussation of the oculo motor nerves, and therefore the eyes and the extremities are paralyzed on the same side. Therefore, the inclination of the eyes is

of diagnostic importance in locating the situation of an apoplexy.

If the eyes turn toward the paralyzed side, we have an alternating paralysis, and the lesion is in the pons on the opposite side. If the eyes turn toward the sound side, the hemorrhage is in one or other hemisphere above the pons, and on the same side toward which the eyes turn.

A deviation of the eyes from the side of a cerebral lesion may exist from simple conjugate spasm of the ocular muscles, owing to an irritation in the vicinity of the visual sphere in the occipital cortex of the opposite side, thus simulating the condition found in diseases of the pons. The unimpared ability to move the eyes in the opposite direction will demonstrate the absence of paralysis, and the limbs on the side toward which the eyes are turned will also be in a rigid, spasmodic condition, in contrast with the flaccid paralyzed state.

Another indication of disease of pons is an associate paralysis of the eyes. This differs from the conjugate paralysis just referred to in that the paralysis of the affected muscles is not complete. In conjugate paralysis toward the left, for instance, both eyes are turned to the right, and cannot be moved at all in the opposite direction. In associate paralysis, on the other hand, they can be turned to the left as far as the middle line, but no farther. Such cases have been occa-

sionally observed, but no satisfactory explanation of their cause was offered until, in 1879 or 1880, Duval discovered a bundle of fibres proceeding from the nucleus of origin of the abducens to the oculo-motorius of the opposite side. Later, a case of associate paralysis was reported by Ferreol, in which an autopsy disclosed a small tumor in the immediate vicinity of origin of the sixth nerve. Stellwag saw this peculiar condition in a case of Basedow's disease. Gowers locates a lesion causing such associate paralysis in the tegmental region of the pons on one side, above and adjacent to the nucleus of the sixth nerve. If the nucleus of the sixth nerve were involved, the palsy of the external rectus would be complete, so that that eye could not be turned outward at all, even as far as the middle line. Inasmuch also as the internal rectus receives special direct innervation from the third nucleus, it is not incompatible with associate paralysis, that contraction of the impaired internal rectus should be retained in convergence of the visual axes, or that this eye should turn in connection with its fellow, whose external rectus is impaired, as far as the middle line in the direction of the associate paralysis.

Two other varieties of this associate paralysis in horizontal movement have been described, the one by M. Parinaud, in which spasm of the internal rectus exists, and the other by Sauvineau, in which spasm of the external rectus exists. These differences are explained

by a slight variation in the situation of the lesion, so that with the paralysis for lateral associated movement there is an irritation of one or the other nerve. Thus both associate and conjugate paralysis point to a lesion in the corresponding side of the pons. There is some reason to believe that the pons also contains centres which preside over the associated lateral and vertical movements of the eyes.

"Paralysis of both upward and downward movements of the eyes, bilateral and accompanied with ptosis, has been observed with disease of the corpus striatum and optic thalamus. Loss of conjugate movement upwards has been caused by a lesion of the tubercula quadrigemina." (Gowers).

Verray reported such a case accompanied with tendency to deviate toward the left when walking. The affection came on suddenly and was attributed to a small hemorrhage in the tubercula quadrigemina. See "*Révue Med. de la Suisse Romande*," March, 1893.

"Beckheim believed that the superior olive (which extends the whole length of the pons) acted as a reflex centre for correlating movements of the head and eyes with auditory impressions, and its lesion might therefore be expected to interfere with such correlations, causing a lack of response by the head and eyes to sounds coming from various directions." (Chas. K. Mills in "International Clinics," October, 1895). Such a centre for reflex movements of the head and

eyes in connection with auditory impressions has been referred to the internal geniculate body.

NUCLEAR PARALYSES.

A nuclear paralysis may affect one or both eyes, but, owing to the free communication between the nuclei of the opposite sides, the latter is more frequent. As has been previously mentioned, the motor-oculi arises from separate nuclei in the aqueduct of Sylvius. Hence an isolated paralysis of one or more of the branches of the nerve, or their successive implication, points unmistakably to a nuclear lesion. Not infrequently, the intra-ocular muscles escape, because their motor fibres receive a separate vascular supply. A total paralysis of all the muscles of both eyes is, in all probability, of nuclear origin. The only other explanation is a separate lesion involving each cavernous sinus or each optic foramen.

CORTICAL OCULAR PARALYSES.

The course of the nerve fibres between their nuclei of origin and the cortical visual centres has not been anatomically demonstrated, and we have no data for differentiating ocular paralyses due to lesions in these two localities, i. e., cortical or fascicular. They are always binocular and conjugate. Each visual area not only receives impressions from the opposite half fields of

vision of the two eyes, but also presides over the voluntary, conjugate movements in the same direction. An irritation at a given point of the occipital cortex, as has been mentioned, causes deviation of both eyes toward the particular part of the opposite visual field which is in anatomical relation with that spot (conjugate spasm), and a circumscribed lesion results in hemianopic visual defects and corresponding loss of conjugate movement. The visual centre on the right side is associated with the left half fields of vision and with conjugute movements toward that side, and vice versa. Therefore the lesion is always on the side opposite the paralysis, and the eyes deviate toward the side of the lesion.

There are two features which are characteristic of cortical ophthalmoplegias, and which serve to distinguish them from similar motor defects due to lesions of the pons.

First, involuntary and reflex movements are preserved. If a strong light is thrown into one or both eyes from the paralyzed side, they will turn in the direction of the light, that is, in the direction in which voluntary movement is lost. The same occurs under the impulse of a sudden command or a loud noise. Such involuntary movements are absent in all cases where the lesion is at or below the nuclei.

Second, cortical paralyes are associated with visual disturbances and pupillary conditions which are of great value in locating the causative lesion, but a dis-

cussion of such symptoms will be reserved for a subsequent chapter.

Dissociate paralyses, that is, of individual muscles not concerned in associate movements, are also of central origin.

In muscular affections due to central disease there is very great difficulty in overcoming the resulting diplopia. Even with carefully selected prisms, the patient finds it almost impossible to unite the double images.

Having said so much in regard to the localization of a lesion, let us now inquire whether the eye symptoms furnish any evidence of the nature of the diseased process. We cannot state from the presence of ocular palsy alone that there exists hemorrhage, embolism, thrombosis, meningitis, tumor, neuritis, effusion of lymph serum or pus, softening or sclerosis, any one of which may be the cause of the existing symptoms, but still these same ocular palsies do often point toward the nature as well as the location of the central lesion.

A sudden lesion, developing immediately or within a few hours and causing paralysis, is always a vascular one, either a hemorrhage or an occlusion. An acute lesion, developing in a few days or weeks, is probably inflammatory. A chronic one indicates degenerative processes or gradual pressure from a neoplasm.

Ocular paralyses in children, without obvious cause, is to be regarded as a very serious premonition of tubercular meningitis. Such a case occurred not

long since in my practice. I was consulted simply for the secondary squint. I declined to operate, and gave a guarded prognosis, and within a few months the child died with unmistakable signs of this affection. Such symptoms are much less frequently associated with simple meningitis.

In adults ocular paralyses are very suggestive of cerebral syphilis. Von Graefe says that one-third of all ocular paralyses are due to syphilis. The proportion has been estimated as high as one-half. They occur usually in the later stages of the disease. The third nerve is affected in three-quarters of such cases and the sixth in one-quarter. It may be a nuclear lesion, a neuritis of the trunk or root of the nerve, or it may be compressed in its course along the base of the brain by a gumma. Such a paralysis may be the initial symptom of cerebral syphilis. One variety, where several muscles of both eyes are affected successively and transiently, varying in degree and duration, is considered almost pathognomonic of the cerebral form of this disease. Anaesthesia or dysaesthesia of the skin of the face frequently accompanies syphilitic ocular palsies.

Ocular palsies with similar manifestations are also very frequent in locomotor ataxia, or tabes dorsalis. They may be an early or late feature of the case. Frequently, as in one case coming under my observation, they preceded for some time the other developments of the disease. Mott in "International Clinics," 1895, Vol.

I, page 127, says that 25 per cent. of the cases commence with ocular paralysis. Two varieties are considered suspicious, viz., "insignificant paresis which easily recovers, but soon reappears in one or the other eye. In other cases there quickly becomes associated with a perhaps trifling paresis of the abducens, a contraction of the internal rectus." (Mauthner's "*Gehirn und Auge*"). Such cases are very obstinate to treatment.

It is estimated that from 20 to 40 per cent. of the cases of tabes are accompanied with ocular palsies, which are of sudden development and of transient duration with frequent relapses. One or both eyes may be affected. Thus we see that the symptoms resemble very closely those resulting from syphilis, and it should be remembered that most tabetic patients have at some time been syphilitic.

Authorities differ as to the order of frequency of the involvement of the muscles. In 100 cases of tabes, Dillman found paresis of one or more of the muscles supplied by the third nerve 26 times; abducens paresis 12 times; trochlearis paresis 3 times. Kahler, on the contrary, considers paralysis of the external rectus of most frequent occurrence and of the levator palpebrae next in frequency.

Associate paralysis of convergence has been noticed. Knies says: "Every paralysis of an ocular muscle which occurs suddenly in a healthy person (without injury, apoplexy, or other brain symptoms,

diabetes, mellitus or insipidus, syphilis, albuminuria, etc.) arouses the suspicion of a beginning tabes, especially if it recovers in a comparatively short time or subsequently relapses."

Thus in the majority of ocular paralyses, especially in adults, we shall find the exciting cause to be either syphilis or tabes, and the following points of differential diagnosis between these two affections as formulated by Fournier in the "*Bull. de Med.*," 1887, is of value, although not of universal application:

Tabes.	Syphilis.
Often only a single muscle involved (nerve roots chiefly).	Larger number of muscles involved and accompanied with headache, vertigo, epileptic attacks, aphasia, mental disorders, etc.
Accommodation intact for a long time.	Accommodation suffers early.
Often temporary, sometimes lasting but a few hours.	More permanent and develop more gradually.

Galezowski, on the other hand, says that unilateral paralysis of accommodation without mydriasis is often the first sign of beginning tabes, and is associated with anaesthesia of patches of skin in the temporal region. Those which occur early in the disease more frequently recover than those developing later.

Multiple sclerosis and general paresis also give rise to ocular paralyses of similar character. Other symptoms will be present to establish a differential diagnosis. A paralysis of both external recti has been noticed as an early manifestation of the latter affection.

Disease of the cerebral vessels, resulting from diabetes and renal affections, may be the cause of ocular paralysis, and hence an examination of the urine should not be neglected when a satisfactory explanation has not been reached.

Ocular paralyses may be rheumatic and may affect a single muscle or several. Usually one or more contiguous branches of the nerve are involved, the superior rectus and the levator palpebrae for instance. They follow exposure, are associated with other rheumatic affections, remote or present, and are, as a rule, limited to one eye, whereas ocular paralyses from central disease are usually bilateral. Such cases are generally regarded as of nuclear origin. Diphtheria rarely causes paralysis of the external eye muscles. Such cases, when they do occur, are usually peripheral, and are the sequence of hemorrhages or neuritis, but sometimes they are nuclear. They occur later in the disease and usually slowly recover. An ocular paralysis, due to peripheral neuritis, is most frequently caused by alcohol, and we should expect to find associated symptoms of peripheral neuritis in other localities.

PATHOLOGY OF NUCLEAR PARALYSES.

It has been remarked previously that progressive paralysis of the different branches of the third nerve, or a total paralysis of all the muscles of both eyes, is almost conclusive evidence of a nuclear lesion. This lesion may be of varied nature; for instance, a tubercular formation, or a cyst might exist in this situation and produce the symptoms by pressure. A syphilitic atheromatous degeneration, or an embolism of one of the branches of the basilar artery would explain such a condition.

Certain toxic substances, notably lead, alcohol sulphuric acid, and the fumes of charcoal, have produced nuclear ocular paralysis, but more frequently the resulting eye symptoms in such cases of poisoning are due to peripheral neuritis.

Acute and chronic infectious diseases are also occasionally associated with nuclear ocular palsies. They may complicate divers affections, notably bulbar paralysis, progressive muscular atrophy, tabes and disseminate sclerosis.

There is an acute and a chronic form of nuclear paralysis. An acute form develops in intemperate persons and is usually fatal.

Knies says: "Progressive paralysis of the ocular muscles is an essential feature of polio-encephalitis superior, or inflammation of the floor of the fourth ven-

tricle. (Wernicke was the first to differentiate it). The acute hemorrhagic form is usually due to alcohol. The chronic form is the more frequent. The symptoms are generally bilateral, are irregular in development, and affect both the external and internal muscles. The loss of function may be complete or partial. It is rarely an independent disease. Somnolence is an early and characteristic feature which serves as a diagnostic point in distinction from simple nuclear paralyses."

SPASMODIC AFFECTIONS OF THE EXTERNAL OCULAR MUSCLES.

It has been previously remarked that spasm of one or more of the ocular muscles is caused by an irritation of the same locality whose destruction causes paralysis. Hence no further discussion of the localization of cerebral affections, as indicated by ocular spasmodic affections, is necessary. A single muscle or a group of muscles may be affected by irritation of a nerve at its nucleus, or at its cerebral termination. Cortical spasms are always binocular and conjugate. As has been remarked in discussing paralytic affections, the secondary deviation of the eyes in cases of cerebral apoplexy, indicates the side of the brain affected. The eyes point toward the side of the lesion unless the hemorrhage is in the pons or crus, when the eyes and extremities are paralyzed on opposite sides, and the eyes deviate toward the unaffected side of the brain.

Since muscular spasms are the expression of cerebral irritation, we should expect to meet with them in hyperaemic and inflammatory affections of the brain and meninges. Strabismus is frequent in cerebro-spinal and basilar meningitis. It is not infrequently caused by reflex irritation from the teeth and digestive organs. This will be more specifically considered in the chapter on reflex neuroses. There is one form of spasm which is worthy of special mention. I refer to nystagmus, an oscillating, to and fro movement of the eyeballs, usually in the horizontal plane, and caused by alternate contraction and relaxation of the external and internal recti. This condition may be congenital or developed in early childhood. It then indicates simply a lack of voluntary control of the muscles, which is acquired by the child simultaneously with intelligent vision. As soon as he recognizes objects, he directs the eyes toward them, thereby acquiring the faculty of accurate fixation. It is easy to understand that this faculty will be imperfectly developed if vision is too indistinct to create cortical perceptions definite and strong enough to excite the impulse of adjustment. Hence opacities of the cornea due to ophthalmia neonatorum, and congenital cataract, coloboma of the iris and choroid, microphthalmus and high degrees of refractive errors are among the recognized causes of congenital or infantile nystagmus. If both eyes are imperfect, the nystagmus is constant, but if one eye only suffers, the tremor is only noticed when an effort

is made to fix an object with this eye, but then it is bilateral. It may also result from arrested development of the nerve trunks or cortical centres, so that the motor stimulus is wanting though visual impressions are accurately received and interpreted.

Nystagmus also develops in persons who use their eyes for long periods with insufficient light, as the so-called "Miner's Nystagmus." The confinement in dark, ill-ventilated, underground chambers, and the breathing of poisonous gasses, added to the strain upon the eyes, consequent upon working with insufficient light and keeping the eyes fixed in an unnatural position many hours a day, which the nature of the work necessitates, are the causes of this affection. Night blindness often develops simultaneously.

When nystagmus develops in adults, who are not engaged in such occupations, it is a valuable diagnostic sign indicating the existence of serious organic disease. But it affords no evidence of the location or the nature of the disease, for it has been found associated with meningeal and subdural hemorrhage, pachy-meningitis-hemorrhagica, in tubercular basilar meningitis, cerebral hemorrhage, thrombosis in the sinuses, tumor, softening, disease of the optic thalamus, lesions of the cerebellum, in disease of one side of the pons, and in degenerative diseases of the cord. Gowers, in the last edition of his "Diseases of the Nervous System," says: "The practical significance of nystagmus is extremely great, not

from any distinct indication of the seat, or precise nature of the disease, but because it shows the presence of *more than functional disturbance.* It is often marked in the early stage of degenerative disease when other symptoms are equivocal, and a search for it should never be omitted, and should include always the upward movement of the eyes. It may be trusted without hesitation, and in a large number of cases prevents a mistake in diagnosis."

It occurs very frequently with multiple sclerosis and is a characteristic symptom and a valuable diagnostic sign. It is said to occur in 12 per cent. of all cases of this disease. When tremor of the eyes on fixing an object is associated with true nystagmus, we have an additional indication of multiple sclerosis. According to Knies, the symptom is caused by sclerotic foci in the vicinity of the muscle nuclei, whereby the conduction is interfered with, but not abolished. In other cases it indicates incomplete motor paralysis of the cerebral cortex. It rarely occurs in tabes and paralysis agitans.

"Charcot regards nystagmus-like twitching of the eyes, that is, irregular movements when an object is fixed, as a valuable diagnostic sign of Friedreich's disease or hereditary ataxia, which begins generally at the period of puberty." (Knies).

In opposition to the views of Gowers, above expressed, that the presence of nystagmus always indicates an organic nervous disease, Dr. Sabrazes, in "*Semaine*

Medic," for September 26, 1894, and quoted in the English edition of "*Annal d'oculistique*," says:

"Is acquired nystagmus, the diagnostic value of which I am about to sketch, always produced by a permanent organic lesion? May there not be cases in which the successive elimination of the many causes cited to explain nystagmus, lead the observer to suspect the possibility of a purely functional disturbance arising from hysterical phenomena?

"This is not the opinion of neurologists, and particularly of Charcot, who said, in 1892, in the course of a lecture on multiple sclerosis and its ocular phenomena: 'Nystagmus is never found in hysteria or in tabes.'

"But the existence of a single indisputable case, carefully observed, suffices to controvert this too strong assertion and to show that an hysterical nystagmus may really exist.

"The author confirms his assertion by reporting a case of manifest hysteria where the nystagmus was independent of any lesion of the visual apparatus. Furthermore, hypnotic suggestion caused the disappearance of all symptoms of disease as soon as it was practiced.

"Nystagmus should not, then, be taken as an absolute proof of multiple sclerosis without considering the possibility of hysteria." (Valude).

CHAPTER III.

AFFECTIONS OF THE LENS AND IRIS. BEHAVIOR OF THE PUPIL AND OF THE ACCOMMODATION.

AFFECTIONS OF THE LENS.

The lens derives its nourishment from the arteries of the ciliary processes. A lenticular opacity is always an evidence of impaired nutrition and there are two varieties of cataract which are of special diagnostic significance.

I. A laminated cataract sometimes occurs in young children in consequence of convulsions, and the latter may be caused by rachitis. An explanation has been offered upon the supposition that a spasm of the ciliary muscle accompanies the general convulsions, and thus interferes with the circulation in the arteries of the ciliary processes. The development and growth of the lens continues up to the sixth year, and those parts which have been completely formed previous to the occurrence of the convulsions remain transparent, while the nutrition of the portion which is still in a formative stage suffers and hence is more or less cloudy. The opacity does not develop immediately, but within a few days or weeks subsequent to the convulsion. There-

fore the occurrence of a laminated cataract indicates the necessity for constitutional treatment, especially with reference to rachitis.

II. A soft or cortical cataract in older children or young adults is frequently caused by diabetes, owing to the absorption of water and to the presence of glucose. Both eyes are usually affected. Therefore the urine should always be examined in such cases.

La Grange ("*Arch. d'Ophthal.*," 1887, Jan.) found 13 cases of cataract among 52 diabetics, and Galezowski ("*Jahr. f. Aug.*," 1883, p. 297) found 46 cases of cataract in 144 diabetics.

AFFECTIONS OF THE IRIS.

Inflammation of the iris (iritis) always arouses a suspicion of syphilis, and there is one form of the affection which warrants a positive diagnosis of this disease. The proportion of cases of iritis which are of specific nature has been estimated by various writers at from one-fourth, (Arlt), to three-quarters. Probably fifty per cent. is a safe estimate. Not all of them, however, manifest the characteristic feature giving rise to the classification of "gummous," "condylomatous" or "papulous" iritis. This consists in the development of "yellow or dirty orange colored nodules, surrounded by a narrow red zone two or three millimetres in diameter, more often toward the pupillary margin and below." The reddish color of the nodules distinguishes the syphilitic iritis

from the tubercular and leprous iritis, in which there also occurs a formation of tubercles or nodules. The former are grayish red or yellowish white in color, and are not much larger than a pin's head. There are usually several, and they may appear and disappear for a considerable length of time. Ludwig Bach makes the assertion in Knapp's Archives, Jan., 1895, that iritis is just as often of tuberculous as of syphilitic origin, but this is probably an exaggeration. He states also that the ciliary portion of the iris is the part most often affected by tuberculosis. The leprous nodules are grayish in color and are situated towards the outer edge of the iris, and usually grow until they fill the anterior chamber and induce secondary cyclitis and irido-cyclitis, and destruction of the eye. Leprosy is a rare disease in this country, while tuberculosis is common. Probably iritis is never a primary manifestation of either disease, and there will be coexistent symptoms which will render a diagnosis unequivocal.

Syphilitic iritis is most frequently observed in the secondary stage of the acquired form. It has been observed within three weeks after the appearance of the initial lesion. When iritis develops in children, especially in infants, it is usually a manifestation of hereditary lues.

Sarcoma, also, sometimes develops in the iris, producing symptoms somewhat analagous to those of syphilis and leprosy. The color of the growths and the

course of development, the history and concomitant symptoms will decide the diagnosis.

Diabetes is occasionally associated with a form of iritis, with a significant symptom of an unusually extensive exudation, purulent or fibrinous, into the anterior chamber.

Iritis may be idiopathic, and caused by exposure to cold, especially in rheumatic subjects, and it is frequently associated with gonorrhoea. Certain conditions frequently, though not invariably, accompany both of these varieties, and render their differential diagnosis difficult without the aid of other symptoms. Both are prone to relapse, and are accompanied with pain and inflammation of the joints, and both are attended with a thick, fibrinous, easily coagulating exudation, with a tendency to firm adhesion to the lens and to occlusion of the pupil.

Distinguishing features of the rheumatic form are the greater severity of the pain, the aggravation from moving the eye, and the rarity of suppuration.

It is stated that iritis often complicates relapsing fever, and, while of no diagnostic importance, the fact should be borne in mind that the affection may be promptly recognized and correctly treated.

Finally, in cases of iritis without obvious cause, the urine should be examined, for it has been ascribed to albuminuria.

BEHAVIOR OF THE PUPIL AND OF THE ACCOMMODATION.

Before discussing the significance of pathological conditions of the pupil, a few words regarding its natural form and size, and the methods of examination are desirable.

The pupil is the nearly round opening in the muscular curtain of the iris. It appears black under ordinary circumstances because the part of the retina presenting through it is not illuminated. The iris is composed of both radiating fibres whose contraction enlarges the opening, and of constrictor fibres by means of which the opening is made smaller. The size of the pupil therefore varies considerably under physiological conditions. It contracts under the stimulus of light, upon efforts of accommodation, and with convergence, and it dilates under feeble illumination, and upon irritation of the skin, especially of the face and of the back of the neck. It is larger in children and smaller in old age than in adult life. It is also slightly larger in myopia and smaller in hypermetropia than in emmetropia. The diminution of accommodation in the former condition, and its increase in the latter is the explanation of the varying size.

Its diameter, under ordinary illumination, is from 2.5 mm.–5.8 mm., with an average of about 4 mm., and a constant departure from this standard is usually an indication of disease. The pupils of the two eyes should be

of uniform size, although very careful examination frequently demonstrates trifling variations not apparent upon ordinary inspection, and they should react consensually and equally to stimuli applied to either eye singly, or to both together. When only one pupil is illuminated, for instance, both should contract to the same extent. If any inequality is noticed, the one showing the lesser reaction is usually the pathological one, but this is not invariable.

The behavior of the pupils is of very great practical significance in the diagnosis both of the location and nature of nervous diseases, and therefore its examination should be conducted systematically and with great care. The following method is recommended: It is assumed that any inflammation of the iris or posterior synechiae is absent, and that the media are clear. The patient being seated before a window with moderate illumination, the size and form of each pupil should be separately measured with a rule or, preferably, with a pupillometer constructed especially for this purpose, while he is gazing straight before him at a distance. The observation should be made when both eyes are open, and when they are alternately closed or shaded, carefully noting any movement of contraction or dilatation, and any lack of uniformity in such movements in the two eyes. The reaction upon convergence and accommodation is now studied by fixing the patient's gaze upon the examiner's finger, which is gradually ap-

proached along the middle line to about 8 inches in front of the patient's eyes. To study the accommodative reaction independently of convergence, each eye should be examined separately. The one under observation should be directed straight forward and the other one closed. After a few seconds he is made to suddenly fix his gaze upon some near object, without altering the direction of his vision. The pupil should immediately contract, and dilate again as soon as the object is removed.

The light reflex is best observed by concentrating a pencil of light, preferably from an artificial source of illumination, upon the pupil by means of a convex lens, and watching the resulting contraction of each pupil. The eyes are to be separately tested, the one not under observation being shielded from direct exposure to the light, but not closed. The light should be thrown into the eye from different directions, and its intensity varied to test the sensibility of different portions of the retina and the degree of such sensibility. These tests should be several times repeated in obscure cases, when the result of the first examination is not conclusive, and it is often advisable to place the patient in a darkened room for some minutes previously.

Having discussed the methods of examination, I pass to what may be learned from the results so obtained.

Dilatation and contraction of the pupils may be due to direct stimulation of the motor nerves, but they are usually reflex acts. The light reflex, the skin reflex, and the contraction attending efforts of accommodation and convergence have been already mentioned.

Mydriasis and myosis have no special significance as isolated symptoms, but, taken in connection with other symptoms, they are sometimes an index of the nature and location of a morbid process, and the different forms of paralysis of the iris muscles are very suggestive diagnostic indications, as will be apparent in a later discussion.

If we recall the paths which the afferent and efferent impulses traverse in each instance of reflex and associated action, we shall more clearly understand the morbid processes which abolish these acts.

1. CONTRACTION OF THE PUPIL.—Contraction of the pupil under the stimulus of light (the light reflex) is accomplished by an afferent impulse passing from the retina along the optic nerve and optic tract to the thalamus, and by way of the habenular ganglion to the pineal gland. From each of these localities (the habenular ganglion and the pineal gland) fibres pass to the oculo-motor nucleus in the aqueduct of Sylvius, and from thence the motor impulse travels along the third nerve to the ciliary ganglion, and through the ciliary nerves to the constrictor fibres of the iris.

The consensual reaction of the pupils is explained by the existence of communicating branches between the habenular ganglia in either hemisphere of the brain, the communication taking place through the posterior commissure, and also by the decussation of the optic nerve fibres in the chiasm, so that stimulation transmitted from the retina of one eye reaches the visual centres through the medium of both optic tracts. (Some writers locate the reflex centre from the iris in the anterior corpora quadrigemina instead of the habenular ganglion).

It is evident that the pupillary light reflex will be lost whenever the conduction fails either in the optic or the third nerve.

Accommodation, or the adjustment of the vision for near objects, is effected by contraction of the ciliary muscle, which, as well as the iris, is under control of the third nerve. An act of accommodation is usually associated with contraction of the pupil. It should be remembered that this is, to some extent, a mechanical result of the contraction of the ciliary muscle whereby a larger amount of blood is forced into the iris. Both the reflex and associated contraction fail simultaneously, as a rule, but it is possible for the latter to be preserved, when the light reflex fails, constituting the so-called "Argyll-Robertson pupil," the significance of which will be referred to later.

The reflex arc by which the pupillary reaction attending convergence of the visual axes is accomplished is probably completed by fibres which pass from the optic tract to the corpora quadrigemina, and from thence to the third nucleus. The function of the corpora quadrigemina is not yet positively determined, but it seems almost certain that they preside over "ocular movements in their relation to visual impressions." (Gowers).

2. Dilatation of the Pupil.—Dilatation of the pupil results passively from relaxation of the sphincter pupillae, and actively from the contraction of the dilator fibres, through stimulation of the sympathetic nerves which govern them. In this manner direct excitation of the cilio-spinal centre in the upper dorsal portion of the cord produces mydriasis, and irritation of the skin has also this effect, by reflex action. In the latter case, the afferent impulse reaches the corpora quadrigemina through the spinal cord, and the motor impulse is thence transmitted again through the upper cervical cord by way of the seventh and eighth cervical and first dorsal nerve roots, to the cervical sympathetic, thence to the cavernous plexus, ciliary ganglion and ciliary nerves. Some of the sympathetic fibres reach the iris by a different path, as is proven by the fact that irritation of the trunk of the sympathetic in the neck produces dilatation of the pupil after removal of the ciliary ganglion. The ophthalmic division of the

fifth nerve is one of the channels for such sympathetic influence.

Thus the skin reflex will be lost whenever the conduction fails in either direction, in consequence of a lesion in the upper cervical region of the cord, in the cervical sympathetic, or along the path between the latter and the ciliary ganglion. When the light reflex is lost in diseased conditions, the skin reflex generally fails also, but not always. The skin reflex may be preserved when the light reflex is lost.

As of diagnostic significance in connection with nervous affections, we recognize a spastic and a paretic variety of both mydriasis and myosis, and, in a given case, it is desirable to inquire which variety exists.

Mydriasis—The spastic variety of mydriasis is caused by contraction of the radiating fibres of the iris through stimulation of the sympathetic motor nerves. The widely dilated pupil, in such conditions, still contracts with efforts of accommodation and upon the stimulus of light, showing that the constrictor fibres and the path of the light reflex are unaffected, but there is diminished or absent contraction from the use of myotics such as eserine and pilocarpine.

Other signs of sympathetic irritation are also present, such as dilatation of the palpebral fissure with impaired power of closing the lid. Often, also, careful observation shows slight protrusion of the eye, and lack of uniformity in the downward movement of the lid and

eyeball. Those points will enable one to differentiate the spastic from the paretic variety of mydriasis.

The characteristic feature of the latter condition is absence of contraction of the sphincter muscle, manifested especially by loss of the light reflex. It presents a moderately dilated and immobile pupil in contrast to the widely dilated and sensitive pupil of spastic mydriasis. Another feature of paretic mydriasis is that it can be increased by atropin, which, when dropped into the conjunctival sac, has the two-fold action of paralyzing the terminal fibres of the third nerve and of stimulating the sympathetic.

It has been previously stated that the light reflex will be wanting when the conduction fails either in the optic or the third nerve, hence we might subdivide paretic mydriasis into (a) sensory or centripetal, and (b) motor or centrifugal paresis. With the former, the mydriasis is always bilateral and associated with more or less loss of sight, so that the visual perception is too faint to excite the motor impulse. The latter form is usually associated with loss of function of one or more of the extrinsic eye muscles, and indicates an interruption of the motor impulse at some point. It may exist with unimpaired sight.

Having determined in a given case which variety of mydriasis is present, we are prepared to inquire next, what deductions can logically be drawn therefrom?

I. Spastic mydriasis may affect one or both eyes, and indicates, as has been mentioned, an irritation of the cilio-spinal centre situated in the lower cervical and upper dorsal region of the cord, or of the cervical sympathetic. This irritation may be direct or reflex.

Direct irritation exists in the early stages of all inflammatory diseases of the spinal cord and its membranes in this locality, such as spinal congestion, meningitis, and neoplasms, and in the so-called "spinal irritation," which is frequently met with in nervous and chlorotic young women. It is also a premonitory sign of tabes.

Reflex irritation results from irritation of the skin (the so-called skin reflex) already referred to. It accompanies extensive cutaneous burns and severe urticaria. Pinching the skin at the back of the neck causes mydriasis in certain cases of meningitis, and is known as Parrot's sign. Intestinal worms cause spastic mydriasis by reflex action; also disease of the apex of the lung, and aneurisms of the aorta and arteria innominata.

In the latter cases the mydriasis is on the side of the lesion. Cerebral affections with increased intracranial pressure, and certain mental states, fright, acute mania and melancholia, for instance, are, at times, associated with this form of mydriasis, the explanation of which must be a reflex irritation communicated to the dilating centre.

Spastic mydriasis, then, indicates the locality of the casual affection, but furnishes no definite information as to its nature. Sometimes dilatation of one pupil with preserved reaction to light, simulating spastic mydriasis, is observed, which is due to some peculiar condition of the iris. Such a condition is not of serious import, and may persist for years.

PARETIC MYDRIASIS. LOSS OF THE LIGHT REFLEX.

As was observed when discussing paralysis of the ocular muscles, the third nerve may be implicated anywhere between the globe and its cortical termination, and the same is equally true of the optic nerve, so that both the motor and sensory varieties of paretic mydriasis may be advantageously classified in the same manner that was adopted with paralysis of the external muscles, namely; 1st, Peripheral; 2d, Intra-cranial (a, Basilar; b, Nuclear); 3d, Cortical.

With the first classification, that is, cases of peripheral origin, whether motor or sensory, the present discussion does not concern itself, as they have no relation to the diagnosis of nervous diseases. Care should be taken, however, not to confound the symptoms caused by the use of mydriatics, such as atropin, duboisine, cocaine, hyoscyamine, etc., with similar accompaniments of cerebral affections. It is important to remember that all drugs which dilate the pupil also impair the power of accommodation and *vice-versa*. The

same is true of myotics and spasm of the ciliary muscle.

Reflex iridoplegia (it has been previously stated that the pupillary functions are usually reflex) is ordinarily bilateral.

Loss of the light reflex, when monocular, may be either peripheral, basilar or nuclear, and of itself does not furnish data for exact localization. It is always motor. Otherwise contraction would follow light stimulus transmitted from the other eye.

Monocular mydriasis is sometimes idiopathic, caused by exposure to cold, although in suspected disease of the nerve centres, monocular mydriasis and paralysis of accommodation is to be regarded with suspicion, as suggesting the approach of general paralysis or of insanity.

Wernicke's sign, or "hemianopic pupillary inaction," is a trustworthy guide in the diagnosis of the situation of a lesion causing hemianopic visual disorders. If light thrown upon the blind portion of the retina causes contraction of the pupils, it demonstrates that the cause of the lesion is above the reflex path for the pupillary response to light stimulus.. The converse, namely, lack of contraction of the pupils, is conclusive evidence that the lesion is at the optic thalamus, or between it and the optic nerve stem.

When hemianopic pupillary inaction exists without concomitant hemianopic visual defect, it indicates that the lesion is between the nucleus of the third

nerve and the optic tract. This symptom is known as Knies' sign, and when observed is of definite value in localization.

In case of sudden blindness also the presence or absence of the light reflex is of service in determining the location of the lesion causing it, for, as we have seen in the case of hemianopia, if it is due to a cortical disease affecting the higher centres beyond the "belt-line," so to speak, the light reflex will be preserved, while it will be lost in affections of the retina, optic nerve or tract. There are exceptions to this rule, however, for cases are on record where there was sudden blindness from a lesion affecting the nerve trunks in front of the chiasm, in which the pupillary reflex was preserved. Such cases simply indicate that a slighter stimulus suffices for the production of reflex contraction of the constrictor fibres of the iris, than for the perception of light.

An isolated paralysis of the constrictor fibres of the iris without other signs, points to a circumscribed lesion, probably syphilitic, in the anterior part of the floor of the fourth ventricle, affecting only that part of third nucleus from which arise the fibres supplying the iris. It is possible for such a lesion to be manifested by monocular mydriasis, but from the anastomosis which exists between the nuclei of the two sides, such a condition is extremely unlikely.

Cortical paretic mydriasis is bilateral. It occurs with cerebral anemia, in apoplexy and in meningeal hemorrhage, and in alcoholism. When mydriasis succeeds a primary myosis in apoplexy, it is an unfavorable symptom, showing paralysis of the third nerve from increasing pressure. The presence of dilatation or contraction is a valuable point of differential diagnosis between apoplexy and embolism, as the pupils are unaltered in the latter. It is of frequent occurrence during epileptic seizures, and is associated with conditions of increased intra-cranial pressure which compresses the third nerve, such as tumors and hydrocephalus. It frequently attends the later stages of tubercular meningitis, but is rare in the epidemic cerebro-spinal variety. This fact may be of value in a differential diagnosis. It occurs also in diseases of the cerebellum. In concussion of the brain we find a sluggish action of the pupils without marked dilatation or contraction. In coma from compression, we find one or both pupils dilated and sluggish.

Care should be exercised to discriminate between loss of vision from cerebral affections and the indistinctness resulting from impaired accommodation, or because of the circles of diffusion formed on the retina in consequence of the dilated pupil. In the latter case, *near* vision will be restored by a suitable convex lens when the accommodation is at fault, and the circles of diffusion may be obviated by limiting the amount of light

entering the eye, by looking through a small opening in a card or similar device, thus improving vision for all distances.

Paretic mydriasis is an early symptom of general paralysis and a late manifestation of paralysis of the insane.

Von Graefe called attention to an ephemeral variety of mydriasis, which occurs transiently, at short intervals, and which he regarded as a premonitory symptom of insanity, more especially of "ambitious monomania." It may, however, be of the spastic variety due to spinal irritation. The pupillary phenomena of this disease will be referred to again.

The condition of the pupil is of service in distinguishing an atrophy of the optic nerve resulting from inflammation or caused by cerebral disease, from that form known as spinal atrophy. In the two former cases it is, as a rule, dilated through loss of the light reflex, but in the latter it is contracted, in consequence of paralysis of the sympathetic.

Mydriasis may be a premonition of uraemic intoxication, hence the condition of the pupils should be carefully watched in cases of nephritis when this condition threatens.

Mydriasis with loss of power of accommodation should awaken a suspicion of syphilis, for Alexander states that three-fourths of such cases are of syphilitic

origin, while Uhthoff estimates the proportion due to this cause at one-fourth the whole number.

In cholera, loss of the light reflex is a bad prognostic omen. In the "*Deutsch. Med. Woch.*" for January 23, 1891, Corte states that where the reaction to light fails, a fatal termination is certain, even in an apparently mild case, and, on the other hand, the prognosis is favorable even in very severe cases if the light reflex is preserved, and this he considers true without regard to the existence of a dilated or contracted pupil.

THE ARGYLL ROBERTSON PUPIL.

Usually the reflex contraction of the iris attending efforts of accommodation and convergence is lost simultaneously with failure of the light reflex. It is possible for the associated contraction to be preserved when there is no response to the stimulus of light, and this variety of reflex iridoplegia is known as the "Argyll Robertson pupil," and is a very valuable diagnostic indication in two forms of nervous disease.

The method of testing the accommodative and convergent reaction has already been mentioned on page 59, and need not be repeated here. Noyes maintains that convergence and not accommodation is the factor inducing contraction in the Robertson pupil, and supports his statement by eliminating all accommodation. This he did by placing a strong concave lens before the patient's eye during the examination, and found the re-

sult to be the same. ("Diseases of the Eye," last edition, page 436).

The pupil may be of normal size, dilated or contracted, but usually there is more or less myosis. For this reason it would seem that a consideration of this pupillary condition in connection with mydriasis was inappropriate. But inasmuch as the myosis is only an association, and not an essential feature of the "Argyll Robertson pupil," and since the characteristic of the latter is a paresis of the sphincter iridis, it seems advisable to discuss it here.

Its presence demonstrates a lesion affecting the fibres extending from the tubercula quadrigemina to the third nucleus. Since there is no impairment of vision and no loss of voluntary motion, it is evident that the primary optic ganglia and the third nucleus are unaffected. It is found in locomotor ataxia, or tabes dorsalis, and paresis, or progressive paralysis of the insane, as a characteristic symptom and is very rare in other diseases. Hence it becomes a very valuable aid in diagnosis, especially as it often occurs very early in these two affections. The peculiar mental condition and the disorder of speech in paresis will enable one to make a differential diagnosis. These two diseases may exist simultaneously.

Dillman says that the "Argyll Robertson pupil" is found in 70 per cent. of the cases of tabes, and in 25

per cent. it is a very early symptom. It is estimated that this symptom occurs in half the cases of paresis.

MYOSIS.

With myosis also we recognize a spastic and a paretic variety. The former is the expresssion of irritation of the oculo-motor nerve, and is accompanied by spasm of accommodation. Atropia will cause full dilatation and relaxation of accommodation. It commonly attends hyperaemia of the brain and the early inflammatory period of cerebral affections, and of tubercular and cerebro-spinal meningitis, the irritable premonitory stage of apoplexy and of nacrosis, and the excitement of intoxication from alcohol and chloroform.

We find it with hemorrhage into the cerebral ventricles and into the pons Varolii, in which situations an irritation of the adjacent oculo-motor nucleus results. A neoplasm near the origin or along the course of the third nerve, would, in its early stages, cause spastic myosis, and it accompanies hysterical and epileptic attacks. It may be due to the influence of nicotine, and it occurs in reflex irritation from affections of the fifth nerve.

PARETIC MYOSIS.

The paretic is a much more common form of myosis, and is characterized by failure of the skin reflex, and, when uncomplicated, the pupil is of medium

size and responds both to light and convergence. It is an evidence of paralysis of the sympathetic, which may be due to direct injury of the cervical sympathetic, or to its compression by an anuerism, a tumor, enlarged glands, etc. It is then monocular and is associated with other evidences of sympathetic paralysis, such as slight ptosis and flushing of the corresponding side of the face, with local increase of temperature amounting sometimes to 1.5° F., measured in the nostril or external auditory canal. Such a condition may last for years.

Paretic myosis is generally caused by a chronic disease of the spinal cord. It does not occur, however, when the disease is situated below the upper dorsal region. In this variety of myosis, both pupils are affected, the accommodation is normal, and the pupil can be dilated with atropin, although rather feebly. Atropin has a two-fold action in producing dilatation. It causes paralysis of the terminal fibres of the third nerve distributed to the sphincter-pupillae and ciliary muscle, and at the same time it stimulates the sympathetic which innervates the dilator fibres. This explains the more energetic action of the drug in spasmodic than in the paretic form of myosis. The latter is more especially met with in tabes and progressive paralysis of the insane. Dillman says that it is present in 23 per cent. of the cases of tabes.

We have seen that in these two diseases the "Argyll Robertson pupil" is also usually present, so

that we find the characteristic feature to be that of reflex iridoplegia in the full meaning of the term, that is, a failure of all pupillary reflexes, and this condition serves as a valuable point in differential diagnosis between true tabes and the multiple neuritis (of alcoholic origin, usually) which so closely simulates it, for in the latter there is an absence of myosis and reflex iridoplegia.

In tabes, the reaction to light generally fails first. Knies says: "Absence of pupillary reaction to light, which is followed by loss of reaction to accommodation and to convergence and to cutaneous irritants, is one of the most characteristic signs of impending or beginning tabes, and forms an integral part of the further clinical history of the disease."

"The presence of oculo-pupillary and vaso-motor symptoms of paresis or irritation are important in an accurate local diagnosis in injuries of the spine. Almost every severe spinal injury is attended with vaso-motor paralysis and rise of temperature upon the side of the motor paralysis."

The presence of myosis and iridoplegia with coma of syphilitic origin, enables us to differentiate it from a similar condition due to uraemia or alcoholism in which there is mydriasis. It is hardly necessary to mention the extreme myosis which occurs in opium poisoning. The absence of myosis would be a counter-indication in suspected *acute* poisoning, but it is

worthy of remark that mydriasis is not uncommon in *chronic* opium habitués.

ANISOCOREA.

By this term is understood an inequality in the size of the two pupils, or an irregular and unequal response to the various stimuli.

Knies says that difference in size of the pupils points to the path between the optic ganglia and the nuclei of the third nerve, as the location of the lesion causing it. "It may be an irritative lesion or a commencing paralysis, according as one pupil is too small or too large. The one which reacts most freely and readily to light, convergence, etc., is to be considered the normal one."

Dillman says that this condition occurs in 34 per cent. of the cases of tabes, and therefore it furnishes still another oculo-pupillary sign of that disease in addition to those already mentioned. In common with myosis and reflex iridoplegia, anisocorea is characteristic also of the early stage of general paralysis of the insane.

Oliver, of Philadelphia, in discussing the pupillary phenomena met with in this disease (in the "Med. News" of November 11, 1893), mentions "Irregularities in size and reaction of the pupils with myosis. Diminished action of mydriatics, marked temporary asymmetries, one pupil being quite small and irregular for sev-

eral examinations, while its fellow was large and ovoid or oval."

HIPPUS.

There is a condition of the iris known as "hippus," characterized by alternate contraction and dilatation of the pupil, which often accompanies nystagmus, and is of similar significance. It must not be confounded with that tremor of the iris that accompanies movements of the eye in dislocation or absence of the lens. Hippus occurs frequently in multiple sclerosis, and in connection with hysterical and epileptic seizures.

Rhythmical contractions of the iris have been noticed in typhoid fever during the stage of cerebral manifestations.

"Rapid alteration of myosis and mydriasis has also been observed in tubercular meningitis." (Knies).

DISORDERS OF ACCOMMODATION.

The ciliary muscle and the iris, being both innervated by the third nerve, usually act together. Spasm of accommodation, as a rule, is accompanied with myosis, and paralysis of accommodation with mydriasis. Remembering, however, that the fibres which supply the ciliary muscle arise from a separate nucleus, we can understand how the accommodation may be lost *alone* by a circumscribed nuclear lesion in the vicinity of the floor of the fourth ventricle (the aqueduct). Loss of accommodation (associated or not with mydriasis, or paralysis

of the extrinsic eye muscles) is the most frequent form of post-diphtheritic paralysis, which is generally of nuclear origin, though sometimes it is due to a peripheral neuritis. This is frequently the only manifestation of post-diphtheritic paralysis, and readily yields to appropriate treatment, while, if unrecognized, it may seriously alarm both physician and patient. It may occur very soon or at a considerable time after disappearance of the throat symptoms. It affects both eyes and occurs more often in children than in adults. Frequently the pupil is unaffected. It may be a valuable diagnostic indication in doubtful cases, for the presence of paralysis of accommodation following a sore throat or a membranous inflammation of the conjunctiva, of the vulva, or on a wound, decides definitely the diphtheritic nature of the case. Bilateral cycloplegia, as an isolated symptom, occurs also in certain diseases of the cord, and may be explained by an extension upward of the disease to the third nucleus.

Noyes enumerates the following diseases which may be complicated with paralysis, more or less complete, of the faculty of adjustment of near vision, viz.: diabetes, trichinosis, cerebro-spinal sclerosis, essential anaemia, masturbation and excessive venery, alcoholism, uterine and syphilitic affections, neuralgia of the dental and other branches of the fifth nerve. While of no special value as a means of diagnosis in these affections, a recognition of the cause of the visual disorder is desirable, as influencing the treatment.

CHAPTER IV.

THE OPHTHALMOSCOPIC APPEARANCES OF THE FUNDUS OCULI, INCLUDING AFFECTIONS OF THE CHOROID, RETINA AND OPTIC NERVE.

For the diagnosis of pathological changes in the interior of the eye, behind the crystalline lens, the aid of the ophthalmoscope is necessary. This instrument is of very great value in the diagnosis of cerebral and spinal affections, as well as of certain constitutional disorders, and every progressive physician should be familiar with its use. The instrument consists essentially of a mirror perforated in its centre, and having a series of lenses, any one of which can be brought directly behind the perforation. The pupil of the patient is illuminated by light reflected from a gas jet by the mirror, and on looking through the aperture, directly in the line of the illuminating rays, the observer is enabled to examine the interior of the patient's eye. Many different patterns of the instrument are to be obtained, but for the use of the general practitioner there is none better than Loring's Student's Ophthalmoscope, a cut of which is given below.

The optic nerve is but an off-shoot from the brain, and the retina but an expansion of the former, so

that we have here a continuity of nervous structure subject to identical morbid processes. Moreover, the optic nerve sheaths enter into the structure of the tunics of the eyeballs on the one side, and are continuous with

the cerebral meninges on the other; and the sub-vaginal spaces are tubular prolongations of the cranial sub-arachnoid and sub-dural spaces. It is easy to understand, therefore, how meningitis may be complicated with peri- or insterstitial neuritis, and how watery or purulent exudations in the cranial cavity may cause distention of the nerve sheaths and induce inflammatory changes in the papilla and retina. Again, derangement of the cerebral circulation is accompanied with simultaneous changes in the retinal vessels, and the ophthalmoscope enables one to recognize hyperaemia and anaemia of the retina, hemorrhage, embolism of the arteria centralis, and the varieties of intra-ocular inflammation which are symptomatic of constitutional affections. Frequently before the sight begins to suffer, the ophthal-

moscope reveals changes at the optic disc, and in a doubtful case we are often enabled by its aid to confirm a diagnosis of disease of the brain, heart, kidneys, etc., and to interpret the true meaning of various nervous symptoms not otherwise of definite signification.

In these pages it is not my intention to discuss the differential diagnosis of the various forms of disease at the fundus of the eye. Such a diagnosis requires special training and skill not possessed by the majority of general practitioners. In pursuing the general plan of this treatise, I shall endeavor simply to point out the significance of certain pathological conditions as determined by ophthalmoscopic examination, assuming the reader to be familiar with the use of the instrument, or that, not possessing the requisite skill and knowledge, he appreciates the importance and value of such a procedure, and will call in the aid of a specialist.

AFFECTIONS OF THE CHOROID.

As with iritis, so also with choroiditis, the most frequent cause is syphilis, so that the presence of either the diffuse or the disseminate variety awakens a strong suspicion of a specific origin, but there are no characteristic features which warrant an absolute diagnosis of syphilis. It occurs in both the acquired and the inherited form of the disease, but is most often met with in the second, third or fourth years of the acquired form. When syphilis can be excluded as the exciting

cause, disorders of nutrition, such as anaemia, chlorosis and scrofula, are to be sought for.

We also recognize a metastatic suppurative choroiditis, which is a manifestation of pyaemia, and occurs most frequently as a complication of puerperal fever. It may be the first or the only indication of pyaemia, and hence may lead to the recognition of a hitherto unsuspected suppurative condition.

Knies says that a "sero-plastic purulent" choroiditis occurs frequently as a complication or a sequence of cerebral meningitis, especially in children. In such cases it is usually monocular, and caused primarily by phlebitis of the ophthalmic vein, although there may be "a coincident deposit of morbific material in the pia mater and the choroid."

A septic choroiditis has been noticed after typhoid and intermittent fevers. Tuberculosis of the choroid occurs with or without tubercular meningitis in the form of "single or multiple whitish yellow or rosy yellow round masses over which the retinal vessels pass. Their outlines are rather hazy. They are very rarely surrounded by a striking pigmented zone, and this is important in making a differential diagnosis. The individual nodules usually grow, coalesce and project more and more into the vitreous. Usually they belong to the terminal stage of tuberculosis, and develop shortly before death." Therefore they possess but little diagnostic value as a rule, but their recognition is important, be-

cause sometimes, as Hangg says in "*Dis. Strasbourg*," 1890, "they may apparently constitute the sole manifestation of tuberculosis for a long time."

AFFECTIONS OF THE RETINA AND OPTIC NERVE.

We can draw no fast lines of differentiation between the diagnostic significance of neuritis and retinitis, as they so frequently coexist and merge the one into the other. In general terms it may be stated that in constitutional disorders pathological changes are more pronounced in the tissues of the retina, while the intra-ocular extremity of the optic nerve is first and most predominantly affected by intra-cranial and spinal affections. It is deemed advisable, therefore, to consider separately the ophthalmoscopic appearances of the back ground of the eye as they relate to systemic and nervous diseases, and first let us inquire what data may thus be obtained for the diagnosis of constitutional diseases and those of remote organs.

Retinitis and neuro-retinitis are found in connection with many and diverse constitutional disorders. Often they are of no special diagnostic importance, occurring simply as complications in the course of well pronounced and universally recognized diseases. As examples may be mentioned their occurrence in typhus and typhoid fever, measles, small-pox, diphtheria, pneumonia and intermittent fever; in disturbances of nutrition such as scrofula, scurvy, purpura and oxaluria, and

in acute anaemia, especially after hemorrhage from the uterus or stomach. Sometimes they occur as a rheumatic manifestation and in menstrual disturbances.

In other instances, affections of the optic nerve and retina are of great value in the diagnosis of constitutional maladies and disorders of remote organs. In general, it may be stated that morbid appearances in these tissues, when not connected with nervous diseases, or indicative of them, point to lesions of the heart and blood vessels, to syphilis or to renal disease.

CHANGES IN THE CIRCULATION OF THE RETINA.

Venous hyperaemia of the retina accompanies cardiac affections in which the return of the blood toward the heart is impeded, as in valvular lesions and in fatty heart. Associated with the above, there may be pulsation of the retinal arteries. This association of arterial pulsation and fulness of the retinal veins almost always exists in aortic insufficiency. The explanation of the former consists in a hypertrophy of the left ventricle, whereby the blood pressure is increased during its contraction, and the venous hyperaemia results from the regurgitation which accompanies the ventricular diastole. The same conditions also explain the alternate reddening and pallor of the disc observed in aortic insufficiency. Retinal pulsation sometimes occurs with aneurism of the aorta and arteria innominata.

An embolism of the central retinal artery may be the first indication of an insidious endocarditis, as was the case in a patient of the writer. Thus the ophthalmoscope may afford the first evidence of heart disease, and aid in the differential diagnosis of its varied forms.

Hemorrhage into the retina, without accompanying inflammation, indicates (a) increased blood pressure, such as occurs in hypertrophy of the heart, and in valvular lesions and in *suppresio mensuum*; (b) disease of the retinal vessels; (c) morbid states of the blood.

Arterio-sclerosis and an atheromatous condition of the retinal arteries not infrequently cause hemorrhage. With the ophthalmoscope the arteries appear narrower than usual, and this narrowing is often more conspicuous at certain portions of the vessel, giving it an irregular wavy outline. These changes are more marked in the region of the macula, because the nutrition of this part of the retina is not equal to that of the other portions. Such a condition of the retinal vessels is of importance as an indication of a similar condition existing in other portions of the body, especially in the brain. When observed, with or without retinal hemorrhages, it is a warning signal of impending cerebral apoplexy, and should lead the physician to adopt precautionary measures to prevent such a catastrophy.

Such vascular degeneration often results from syphilis. It is frequent with renal affections, and it

should also arouse a suspicion of diabetes, for retinal hemorrhages due to vascular degeneration are not infrequent in this condition. They appear, as a rule, in the form of small, roundish spots (not striated). Hirshberg says they always occur when the disease has lasted more than ten or twelve years, and he regards them as of unfavorable prognostic significance. The association of nephritis with the late stages of the diabetes should not be forgotten, and the possibility of the dependence of the retinal hemorrhages upon the latter. With nephritis the hemorrhages are more frequently striated.

Retinal hemorrhages, due to a diseased condition of the blood, occur in infectious diseases, in pyaemia and septicaemia, in profound anaemia, in malaria, in extensive burns of the skin, in poisoning by phosphorus and lead, and after the bites of venomous snakes. Some of these varieties of hemorrhage demand special consideration.

While the diagnosis of infectious diseases is not in any degree dependent upon the existence of retinal hemorrhages, they are not of infrequent occurrence, and are of prognostic importance. They may occur at all stages, and are an unfavorable complication, especially where occurring early, indicating, as they do, a condition of "profound toxaemia." The same may be said of pyaemia and septicaemia. When they occur early in acute septicaemia, a fatal termination may be expected within a very few days. When they are found

after cutaneous burns, they indicate a condition similar to septicaemia, resulting from the absorption of decomposing material.

The presence of retinal hemorrhages affords valuable evidence in the differential diagnosis between chlorosis and pernicious anaemia, for they frequently occur with the latter, but never in pure chlorosis. The hemorrhages are unaccompanied with retinitis, and have frequently a grayish red centre which is considered quite characteristic. The fact that retinal hemorrhages rarely occur in cachectic conditions resembling anaemia is another useful diagnostic point, and emphasizes the importance of ophthalmoscopic examinations in the differential diagnosis of such conditions, especially in severe and chronic cases.

Hemorrhages very frequently attend the various forms of retinitis in which they form an integral factor, and to which we will next devote our attention. For the purpose of general diagnosis we recognize the following varieties of retinitis, viz. :

1. Retinitis albuminurica or nephritica, accompanying different varieties of renal disease, including the retinitis albuminurica of pregnancy.
2. Retinitis syphilitica.
3. Retinitis diabetica.
4. Retinitis leukaemica.
5. Retinitis of gouty origin.

With all varieties the optic nerve is, as a rule,

involved to a greater or less degree, so that the designation neuro-retinitis would define the condition more accurately.

As previously remarked, the differential diagnosis of these varieties of retinitis requires expert skill and experience, and is beyond the scope of this work. The reader is referred to treatises upon ophthalmology for such discussions. The features common to all varieties, but differing in degree and in distribution, are changes in the retinal vessels, congestion, haziness and indistinctness of the outlines of the optic disc, hemorrhages and exudation (serous or plastic) into the retina, sclerosis and fatty degeneration of the vascular and nervous elements, and hyperplasia of the connective tissue. The stellate appearance of the exudation distributed around the macular region in the early stages of the disease is quite characteristic of the nephritic variety of retinitis, although it is, at times, closely simulated by the diabetic, anaemic and leukaemic retinitis. Thus, while there are special features which are more frequently found in certain special varieties of retinitis, there is no variety which is absolutely pathognomonic. It is the presence of retinitis or neuro-retinitis in one of its phases, rather than the special and peculiar features which it presents, that is of special importance to the general practitioner.

Retinitis occurs most often in connection with albuminuria, next with syphilis, and diabetes is consid-

ered the third most frequent cause. We will consider these associations in the order enumerated:

THE RELATION OF RETINITIS AND NEURO-RETINITIS TO ALBUMINURIA.

The ophthalmoscope affords very great assistance in the diagnosis of nephritis. Every oculist of experience is familiar with the fact that retinitis albuminurica is often one of the earliest symptoms of inflammation of the kidneys. Failure of vision, without apparent cause, should lead without delay to an ophthalmoscopic examination, which may disclose the evidence of nephritis, or, in a doubtful case, such an examination, even before there is any impairment of vision, may demonstrate the cause of constitutional symptoms, which previously had not been rightly interpreted.

It has been variously estimated that from 20–33 per cent. of all cases of nephritis are associated with retinitis. It occurs with various forms of chronic kidney disease, but most frequently with the contracted or atrophic kidney. It is rare in the waxy form and in the large white kidney. Next in frequency is the nephritis of pregnancy, and last the post-scarlatinal variety.

Chronic nephritis in all forms is a serious malady, coming on gradually and insidiously. When in an advanced stage it is beyond the curative reach of medical skill, although much may be done to prolong life

and to mitigate suffering. In its early stages it can frequently be arrested and sometimes radically cured. No aids to diagnosis are to be neglected in these conditions, and no warning eye symptom should be misunderstood or disregarded, and I cannot too strongly emphasize the fact that there are changes in the background of the eye, which are almost pathognomonic of disease of the kidneys, and there are others which are highly suggestive of such conditions.

Usually, both eyes are affected, but often in varying degrees. Marple, in the "N. Y. Med. Record" for March 11, 1893, remarks that "According to the testimony of most observers, unilateral neuro-retinitis of Bright's disease, even where it remains unilateral for only a short time, is of rare occurrence. Cases which remain for month or years with only one eye involved are excessively rare. When the affection comes on in a few hours, as after an injury, or remains limited to one eye for months or years, as in chronic renal disease, the ordinarily accepted theories as to its causation seem inadequate. A satisfactory explanation of such cases seems difficult if not impossible."

The ophthalmoscopic appearances bear no definite ratio to the degree of impairment of vision, for preservation of useful sight is not incompatible with pronounced changes in the retina. It is rare that complete blindness results.

The existence of retinitis is not only of diag-

nostic, but of prognostic importance with renal affections. The eye complication may recover, while the nephritis continues. It has been stated upon good authority that "life is rarely prolonged more than one, or, at most, more than two, years," after the development of retinitis.

Bull gives in the "Trans. Am. Oph. So.," a report of the history of 103 cases of chronic renal disease with retinitis. Of these more than 50 per cent. died during the first year, and the majority within six months; 17 per cent. died during the second year.

Those cases associated with retinal hemorrhages are more unfavorable than those without. It has been remarked that the ophthalmoscopic appearances which are so frequently found with renal affections as to constitute a distinct variety, designated as retinitis albuminurica, are also occasionally found in diabetes and leukaemia and pernicious anaemia. They may be present without any assignable cause. Nevertheless, the absence of albuminuria must not be allowed without repeated and careful examinations.

RETINITIS ALBUMINURICA OF PREGNANCY.

Retinitis has been observed as early as the third month, but it is more frequent in the later stages—the seventh or eighth month—and more often in primparae. Since, as has already been remarked, vision is not always affected early in the disease, systematic ophthalmo-

scopic examinations should be made during pregnancy whenever albuminuria exists.

The principal interest which attaches to it is as regards the question of the advisability of the induction of premature labor. Howe, Pooley and Moore have reported cases where this procedure restored the sight.

Howe says ("Amer. Journal Ophthal.," Vol. II, pp. 5, 6, 1885), "The induction of labor is warrantable where retinitis occurs in the early stage of pregnancy, and persists in spite of proper treatment, but is not warrantable in the last few weeks, in spite of the greater ease with which it is accomplished."

Randolph, of Baltimore, concludes from his personal experience that "visual disturbances showing themselves in the last seven weeks of pregnancy, while indicating the same retinal lesion, are of less grave import in so far as sight is concerned, and unless very pronounced and associated with wide-spread ophthalmoscopic changes, should not in themselves call for the induction of premature labor, for literature shows that, in such cases, the sight is completely restored after labor. This is especially true when the retinitis shows itself in the last two weeks of pregnancy." ("Johns Hopkins Hospital Bulletin," Baltimore, June and July, 1894).

The following abstract of a discussion of the subject before the Medical Society of Berlin, January 23, 1895, by Dr. Silex is worthy of record. The report is taken from the "*Annals' d'Occulistique.*"

"All the peculiarities of albuminuric retinitis in pregnancy are not yet well known. Dr. Silex is in possession of preparations which show the vessels intact.

"Its symptomatology and prognosis are very inexactly treated in literature, and its therapeutics have been much neglected. The author has given much attention to this special pathological subject for many years, and has arrived at definite conclusions in regard to the affection.

"To form a judgment of a case, it is necessary first to make a critical ophthalmoscopic examination of the retinal arteries. In the first stages of the disease, the central artery is often seen with the erect image transformed into a large white cord. This is produced by dilatation of the peri-vascular lymphatic space. This change may disappear, but hyaline degeneration of the arteries threatens the canals of the vessels, which may compromise the function of the internal layers of the retina and produce atrophy of this membrane and of the optic nerve. If the vessels are normal, albuminuric retinitis presents little danger. This form of retinitis may arise from an affection of the kidneys during pregnancy, an acute or chronic nephritis, the general symptoms of which are early developed. The visual disturbances develop slowly, most frequently in the second period of pregnancy, especially in primiparae. If they develop rapidly, chronic nephritis must be assumed.

After delivery, vision is established more or less completely.

"In 22 patients examined during a considerable period, 11 recoverd vision more than $\frac{1}{6}$, 10 others remained with a lesser degree of acuity, and five of them were almost blind. This unfortunate result was due to atrophy of the optic nerve, to retino-choroiditis, and to detachment of the retina, which occurred at a later stage in both eyes. These figures are so important, from a social point of view, that Silex advises interference with pregnancy in all cases where retinitis is found. Its prognosis is indeed uncertain; the women are frequently attacked with eclampsia, and the hope of a living child is but faint. In chronic nephritis, danger to the life of the mother and child settles the question of interference; in case of nephritis of pregnancy, premature delivery is not advocated, as in several cases good vision has returned after expectant treatment.

"The condition of the retinal vessels should decide the question of interference. For example, in two pregnant women, at the commencement of the eighth month, one having an acuity of $\frac{6}{18}$, and the other of $\frac{1}{18}$, one might be authorized to perform premature delivery in the first case on account of the vascular changes, while one should wait in the second. It is interesting that Dr. Silex has been able to find large quantities of albumin in the urine during one or two years without development of chronic nephritis in sev-

eral women in whom the retinal lesion had disappeared and the general health had remained good. The long duration of albuminuria has not always a bad prognosis when it can be attributed to a renal affection of pregnancy."

It should be remembered, as Noyes remarks, that "the amaurosis of pregnancy may be independent of uraemia and without changes in the eye grounds, but with a tendency to atrophy of the optic nerve."

RETINITIS SYPHILITICA.

Syphilis is the cause of many cases of retinitis. It occurs in the hereditary as well as the acquired form. It is usually among the secondary symptoms, more rarely a tertiary development. It may occur as a primary affection, or it may accompany iritis or choroiditis or cerebral syphilis.

It presents no distinguishing or pathognomonic features. It usually exhibits the ophthalmoscopic picture of a simple retinitis without marked exudation or hemorrhage. There may be an unusual tendency to the development of connective tissue in the retina and in the vitreous, constituting the so-called retinitis proliferans. A marked tendency to relapse is a somewhat characteristic feature of syphilitic retinitis. A more frequent localization in the central portions of the retina, and a more frequent sequence of optic nerve atrophy are also indicative of a syphilitic origin.

RETINITIS DIABETICA.

Diabetic retinitis is of rare occurrence and indicates a severe type of the disease. It presents many points of similarity to nephritic retinitis, but there are also some peculiarities which the specialist will recognize. In brief, the following points may be mentioned: irregular distribution of the exudation, less frequent implication of the macula, and when in this location, lacking the stellate arrangement, absence of neuritis, and the punctate form of the hemorrhages, which are also of more frequent occurrence. The vascular changes are more marked in the smaller vessels, while the larger ones suffer equally or to a greater extent in albuminuric retinitis. The latter is almost always binocular, while the diabetic form is frequently confined to one eye.

"Every diabetic disease of the retina is of serious diagnostic import, showing degeneration of the vessels, and showing liability to cerebral hemorrhage which is more frequent than in the eye."

RETINITIS LEUKAEMICA.

Retinitis with leucocythemia is not common, and is of no special diagnostic importance. The fundus sometimes presents a peculiar orange hue. As has been previously remarked, it may closely simulate the picture of retinitis albuminurica, and the same is true of the

retinitis which sometimes develops in the course of pernicious anaemia.

GOUTY RETINITIS.

Dr. Bull, of New York, in the "N. Y. Medical Journal" for August, 1893, describes the features of this form of retinitis. He says:

"An arterio- and phlebo-sclerosis of the retina is noticed by ophthalmoscopic examination, and the microscope shows degeneration in the vessels of the choroid and optic nerve as well. A pathognomonic symptom is a peculiar yellowish granular exudation in the retina around the posterior pole of the eye, usually leaving the macula intact, and situated mainly in the nerve fibre layer, although found to some extent in all the layers except the rods and cones. The changes in the fundus are always bilateral, though rarely symmetrical in both eyes. There is marked impairment of central vision, but there is little or no loss of peripheral vision. The loss of central visual perception is progressive up to a certain point, but blindness never results. Hemorrhages are rare, except in the early stages. In the later stages the walls are strengthened by the deposits."

AFFECTIONS OF THE OPTIC NERVE.

In describing the relation of neuritis and atrophy of the optic nerve to intra-cranial and spinal affections, it is proper to remark by way of introduction, that coin-

cident cerebral or spinal diseases and ocular affections do not always or necessarily stand in the relation of cause and effect. A certain condition of the optic nerve revealed by the ophthalmoscope may be idiopathic, or, as we have seen, it may occur as a secondary result of various general and local diseases. Again, habitual or severe headache associated with progressive failure of sight, is not always a sign of brain trouble, but may be due simply to the effort to see distinctly, where the vision is defective from strictly ocular disorders.

Neuritis is very frequently caused by brain disease, especially by tumor, meningitis and abscess. Different authors estimate the proportion of cases of brain tumor in which neuritis exists as from 70 to 90 per cent. Hence, in doubtful cases, a knowledge of its presence or absence is of great value. It usually takes the form of

CHOKED DISC.

The development of a neoplasm in an unyielding skull necessarily causes an increase of the intra-cranial pressure. One result of this increased pressure is a serous and lympathic stasis in the optic nerve with congestion, oedema and swelling of the optic papilla visible with the ophthalmoscope, and constituting choked disc. Another result of increased intra-cranial pressure is a displacement of the fluid contained in the sub-arachnoid and sub-dural spaces. Some of the fluid

would escape through the foramen magnum into the lymph spaces of the cord, while a portion would distend the sheaths of the optic nerve, which, as we have seen, are tubular prolongations of these cavities. Thus the congestion and oedema of the papilla would be increased, and inflammation and exudation and, perhaps, hemorrhage would develop, and the choked disc become a choked neuritis. Both of these conditions indicate, with very few exceptions, an increase of the intra-cranial pressure.

The intensity of the symptoms bears a pretty constant, though not invariable, ratio to the amount of the increased pressure, and as brain tumors cause a greater increase of pressure than any other morbid process, choked disc has come to be considered as almost pathognomonic of the existence of a tumor. It is evident that mechanical distention of the nerve sheaths is not the only factor in causing the neuritis, from the fact that all cases of brain tumor are not associated with it, and also that very small tumors produce it, while occasionally very large tumors exist without it. With chronic hydrocephalus also, in which the intra-cranial pressure is markedly increased, we find as a rule simple atrophy of the optic nerve and no inflammatory changes. "The propagation of tissue irritation" is a recent rather ambiguous expression to explain the *modus operandi* of such an associated condition. It is less frequent with neoplasms of the frontal lobes than with

those situated more posteriorly, especially in the cerebellum. When in one frontal lobe, a unilateral choked disc might be caused, but whenever any variety of neuritis is due to an intra-cranial affection of whatever kind, it is, with few exceptions, bilateral. Hence, we may lay down the rule that a unilateral neuritis is probably *not* dependent upon cerebral disease.

Choked disc is not an early symptom of a cerebral tumor, indeed, a tumor may exist for a long time without causing choked disc. This happens when the tumor is of slow growth. On the other hand, it may occur suddenly in connection with a chronic neoplasm. A *rapidly* growing tumor never causes a *slowly* developing neuritis, although the reverse may be true; that is, a *slowly* developing tumor may take on a *sudden* growth with rapid development of neuritis as mentioned above.

A cerebral abscess frequently induces optic neuritis, but there are no reliable ophthalmoscopic data for arriving at a differential diagnosis between the latter affection and a brain tumor, although we usually find "less swelling and more pronounced inflammation" in cerebral abscess.

Other diseases which cause marked increase of intra-cranial pressure rarely cause true choked disc. Such diseases are tubercular meningitis, chronic hydrocephalus, extensive cerebral hemorrhages and pachymeningitis hemorrhagica. It has also occasionally been found with tumors of the spinal cord, with albuminuria, diabetes, leukaemia and after profuse hemorrhages,

In conclusion it may be remarked that the diagnostic significance of choked disc is extremely valuable, indicating marked increase of intra-cranial pressure, and affording strong presumptive evidence of a tumor as the cause of the increased pressure.

SIMPLE NEURITIS.

The distinction between choked disc and simple neuritis is rather one of degree than of kind, and a brain tumor may be accompanied with a simple neuritis without strong swelling or prominence of the papilla. This is more apt to be the case when the tumor is in the frontal lobe not far distant from the optic nerve. Thus a unilateral neuritis acquires additional diagnostic importance. A true choked disc could hardly develop idiopathically, but simple neuritis sometimes does, after exposure to cold for example, so that we cannot say positively that such a condition necessarily implies the existence of cerebral disease, but it affords strong presumptive evidence of an "irritative process within the skull." A considerable degree of inflammation may exist without impairment of sight, and may be an early symptom before headache, etc., appears. Hence the importance of ophthalmoscopic examination in all doubtful cases is apparent, and in obscure cases without marked mental symptoms, pain or other pronounced indication of cerebral disease, the ophthalmoscope may establish the diagnosis.

With the exception of intra-cranial neoplasms, chronic meningitis is the most frequent cause of neuritis, and it is easy to understand how the inflammation extends in such cases by continuity of structure, so that here we more frequently find an interstitial or a perineuritis. It is found in the majority of cases, and hence is an important aid in differential diagnosis. It does not often occur early in the disease. Acute meningitis, either tuberculous or non-tuberculous, is seldom accompanied with marked ophthalmoscopic changes, nor do we often find them with cerebro-spinal meningitis. Knies, on the contrary, asserts that neuritis is quite frequent in the latter disease.

It has been observed that choked disc may result in consequence of cerebral gummata, but syphilis may attack the nerve of one or both eyes directly, giving rise to simple neuritis, either as an isolated manifestation, or in connection with affections of the cerebral nerves.

Affections of the optic nerve are of value in injuries to the skull, enabling one occasionally to locate a fracture at the orbital foramen or in the immediate vicinity of the optic chiasm.

Similarly, neuritis developing after an injury to the spine, concussion, "railway spine," etc., points to the cilio-spinal region as the seat of the lesion.

Neuritis is sometimes of malarial origin, in which case it is usually bilateral, and it occasionally develops

in the course of typhoid and other infectious diseases.

Dr. White, of Richmond, Va., reports in the "Medical News" for July 15, 1893, a case of optic neuritis occurring in the third week of typhoid, and a second case where optic atrophy was noted four months after an attack of typhoid fever, during the course of which vision had begun to fail, and had continued, notwithstanding the improvement in the general health. He also cited a case of blindness after malarial intermittent.

Gowers says "optic neuritis may follow scarlet fever without any organic change in the brain to cause it, hence the inference that scarlet fever poison has a special action on the nervous system."

Neuritis and retinitis are, at times, complications of dementia paralytica and multiple sclerosis. Multiple neuritis, or pseudo tabes, sometimes occasions an axial neuritis with central scotoma, simulating toxic amblyopia. This is a valuable diagnostic sign between true and false tabes, in the former of which we meet with simple atrophy of the nerve without inflammation. Similar symptoms occasionally result from the influence of gout.

In such cases there may be also chalky deposits along the vessels of the conjunctiva.

ATROPHY OF THE OPTIC NERVE.

The secondary result of neuritis, and especially of choked neuritis, is atrophy, from compression of the

nervous elements by the vascular engorgement and inflammatory exudation. This is called consecutive or secondary atrophy, in distinction from simple atrophy which is not a result of inflammation. The two forms can be differentiated by ophthalmoscopic examination, but a discussion of the points of difference is not within the scope of this treatise. If, in the course of a brain tumor, we have the picture of inflammatory atrophy of the optic nerve, it demonstrates the previous existence of choked disc, and also that the tumor is a chronic condition, because atrophy requires considerable length of time for its development. Thus we see that the condition of the optic nerve is of prognostic as well as diagnostic importance. In exceptional cases a brain tumor causes simple atrophy by direct pressure upon the optic tract, chiasm or nerve. When only one tract suffers compression, corresponding portions of each nerve will show atrophic changes while the remaining halves will be uninjured. When one nerve, between the chiasm and the globe suffers, monocular atrophy ensues. Simple atrophy may result from cerebral meningitis, and it is often an accompaniment of multiple sclerosis, but in the latter disease it is usually incomplete in its development. It occurs late in the course of progressive paralysis, but sometimes it is an early manifestation.

It may also be associated with dementia and idiocy. The most frequent cause of simple optic nerve atrophy is tabes dorsalis, in which disease the atrophy

begins in the outer temporal side. In its fully developed stage it resembles a glaucomatous excavation. It is often an early symptom, and is ordinarily associated with "Argyll-Robertson pupil" and spinal myosis, to each of which reference has been previously made. It may be many years before the essential features of tabes develop. It may occur in any stage of the disease, and the rapid or slow development of the optic nerve affection is in inverse ratio to the motor symptoms, so that it becomes of prognostic as well as diagnostic significance. When the loss of sight is rapid, the locomotor ataxia is usually of slow development and *vice versa*. Sometimes when the atrophy does not appear until the tabetic symptoms are well developed, a temporary arrest of the latter follows the appearance of the visual symptoms. Berger considers the association of diminished sensibility of the conjunctiva and cornea, and false localization of sensation with optic atrophy "a strong presumptive evidence in favor of a tabetic origin of this atrophy."

Both eyes are affected, but not always simultaneously. There may be a considerable interval, perhaps several years, before the second eye is affected. Knies says the "proportion of atrophy with tabes is variously estimated at from 10–35 per cent." Galezowski says, "two-thirds of all optic nerve atrophies are tabetic. Every genuine gray atrophy raises suspicion of tabes." The length of time that elapses between the commence-

ment of the process and complete blindness varies from two months to seventeen years. The average time is three years.

Knies says that in pseudo tabes we find a "grayish-red opacity and obliterated borders of the nasal half of the papilla, with atropic discoloration of the outer halves."

A similar condition is seen in toxic amblyopia.

Acute and chronic myelitis cause neuritis or simple atrophy. The appearance of the optic nerve and retina affords no reliable indication of the amount of vision. Marked deviation from a normal standard may exist for a long time before the sight is noticeably affected; hence the importance of an early ophthalmoscopic examination.

CHAPTER V.

THE SIGHT AND THE FIELD OF VISION. THE SIGNIFICANCE OF VISUAL DISORDERS DUE TO LESIONS IMPLICATING THE INTRA-CRANIAL COURSE OF THE OPTIC NERVE FIBRES, INCLUDING AFFECTIONS OF THE CHIASM, THE TRACT, THE OPTIC GANGLIA, AND THE CORTICAL VISUAL CENTRES, AND PSYCHIC VISUAL DISORDERS.

In the preceding chapters we have discussed the significance of pathological conditions of the various tissues of the eye and its adnexa. We have now to consider certain subjective disorders of vision, irrespective of any ophthalmoscopic changes which may or may not accompany them. Such conditions are of extreme value in the localization of intra-cranial affections, and occasionally in the elucidation of obscure constitutional diseases.

Three methods of estimating the visual acuity are commonly employed. First by the use of test type of different sizes, each of which is seen by a normal eye at a given distance. The amount of vision is then ex-

pressed by a fraction, the numerator of which indicates the distance at which the patient reads the type, and the denominator the distance at which the same type should be read. $V=\frac{20}{50}$, for instance, indicates that at a distance of twenty feet, the patient cannot read print smaller than that which is normally read at fifty feet. His vision is then $\frac{2}{5}$ of the normal standard.

When the sight is too defective to be tested in that way, we ascertain at what distance he can count the fingers of the examiner, and lastly whether he can still distinguish light from darkness.

Blindness, partial or complete, sudden or gradual, temporary or permanent, occurs very frequently in the course of intra-cranial and spinal diseases, and while the mere subjective sensation of failing sight, without ophthalmoscopic findings, affords by itself no basis for the diagnosis of the nature of the causative lesion, it does afford very valuable data for determining the situation of such a lesion. When it affects both eyes, the presumption is warrantable that the cause lies at or behind the chiasm, if we except a simultaneous lesion of both optic nerves, which, though possible, is extremely rare. (Double retro-bulbar neuritis).

One sided blindness, when not dependent upon an ocular affection, must be referred to the optic nerve between the globe and the chiasm, constituting a retro-bulbar neuritis. A discussion of this condition is not relevant in this connection. (See the chapter on toxic

amblyopia). A central scotoma, that is, a dark spot in the centre of the visual field, which is one of the characteristic features of retro-bulbar neuritis, does not, therefore, necessarily indicate a central lesion. It is otherwise with bilateral and symmetrical defects affecting corresponding portions of both visual fields. Such disorders indicate a lesion involving the intra-cranial course of the optic nerve fibres, and a clear understanding of the anatomical path of these fibres is essential for an appreciation of the diagnostic significance of the various forms of hemianopia. It will be profitable, therefore, at this point, to review the course and ultimate termination of the optic nerve fibres, and to enquire into the function of the primary optic ganglia and the cortical visual centres.

The optic tracts from either side unite at the chiasm where they undergo a semi-decussation. The fibres of the right tract are distributed to the right half of each retina, and those of the left tract to the left half of each retina. Each optic nerve thus contains both crossed and uncrossed fibres. The right nerve, for instance, contains uncrossed fibres from the right tract, which are distributed to the right, or temporal side, of the retina, and also crossed fibres from the left optic tract, which are distributed to the nasal half of the right retina. Each optic tract contains the fibres coming from the corresponding halves of the retinae of the two eyes, the right tract those from the right halves, and the left

PLATE I.

tract those from the left halves. The uncrossed bundle of fibres lies at the outer edge of the chiasm on either side.

Plate I illustrates the course of the fibres from either tract to the eye.

Passing backward along the base of the brain, some of the fibres enter the optic thalamus, particularly the posterior portion known as the pulvinar, but the larger part enter the external geniculate body. In both of these bodies the optic nerve fibres are augmented by axis cylinders from their ganglion cells, and then pass upwards to the cortex of the occipital lobe by way of the internal capsule, constituting the optic radiation of Gratiolet.

Thus both the optic thalamus and the external corpus geniculatum are directly concerned with conscious vision, and a lesion of either interrupts the direct line of conduction from the eye to the visual centres. Fibres also pass from the thalamus to the third nucleus, and are concerned with the reflex movements of the iris. A few fibres of the optic tract pass to the anterior corpora quadrigemina, which bodies have no relation to conscious vision, but preside over involuntary conjugate and associated ocular movements dependent upon visual impressions, "and transmit to the visual sphere a knowledge of involuntary movements which follow light stimuli."

Still other fibres pass downward to the posterior columns of the cord and subserve the involuntary reflexes of the general muscular system due to visual impressions from the opposite half of the visual field. By means of these fibres is explained the visual symptoms which occur in the course of locomotor ataxia. A few fibres also pass from the tract to the internal geniculate body and from thence to the posterior corpora quadrigemina, but they are unimportant for our consideration, as these two bodies have no relation with the function of sight. It has been asserted that fibres pass directly from the tract to the visual centres without entering the thalamus or geniculate body, but this has not been positively demonstrated.

Knies considers it probable that a "union of the binocular visual impressions of corresponding halves of the fields of vision into a harmonious single impression takes place in the primary optic ganglia." The harmonious single impression thus formed is thence transmitted to the cortical centre, and thus is exemplified the function of all the basal ganglia, which is to receive, regulate and control impressions from all outside stations, and to transmit them to their respective cerebral centres.

But the optic ganglia have other important functions in presiding over the involuntary movements of the head, neck and extremities, dependent upon visual impressions received from the opposite half of the vis-

ual field, as well as those ocular reflex movements associated with sensory impressions derived from the ear, the face and distant portions of the body.

It is at the visual centre in the cortex of the occipital lobe that visual impressions first awaken conscious perceptions. From this position originate impulses for conscious ocular movements toward the opposite side, and here memory pictures are stored, by virtue of which an object once seen is recognized. The whole area of the occipital cortex is regarded as directly related to the faculty of vision, but the cuneus is the most important portion, and lesions here produce the most pronounced and serious impairment of sight.

The researches of Henschen (see "*Klinische und Anatomische Beiträge zur Pathologie des Gehirns*," Upsala, 1892), seem to indicate that the immediate vicinity of the calcarine fissure is the locality where a lesion causing permanent hemianopia is found.

By means of commissural fibres between the visual centres and the higher intellectual centres visual impressions excite mental processes.

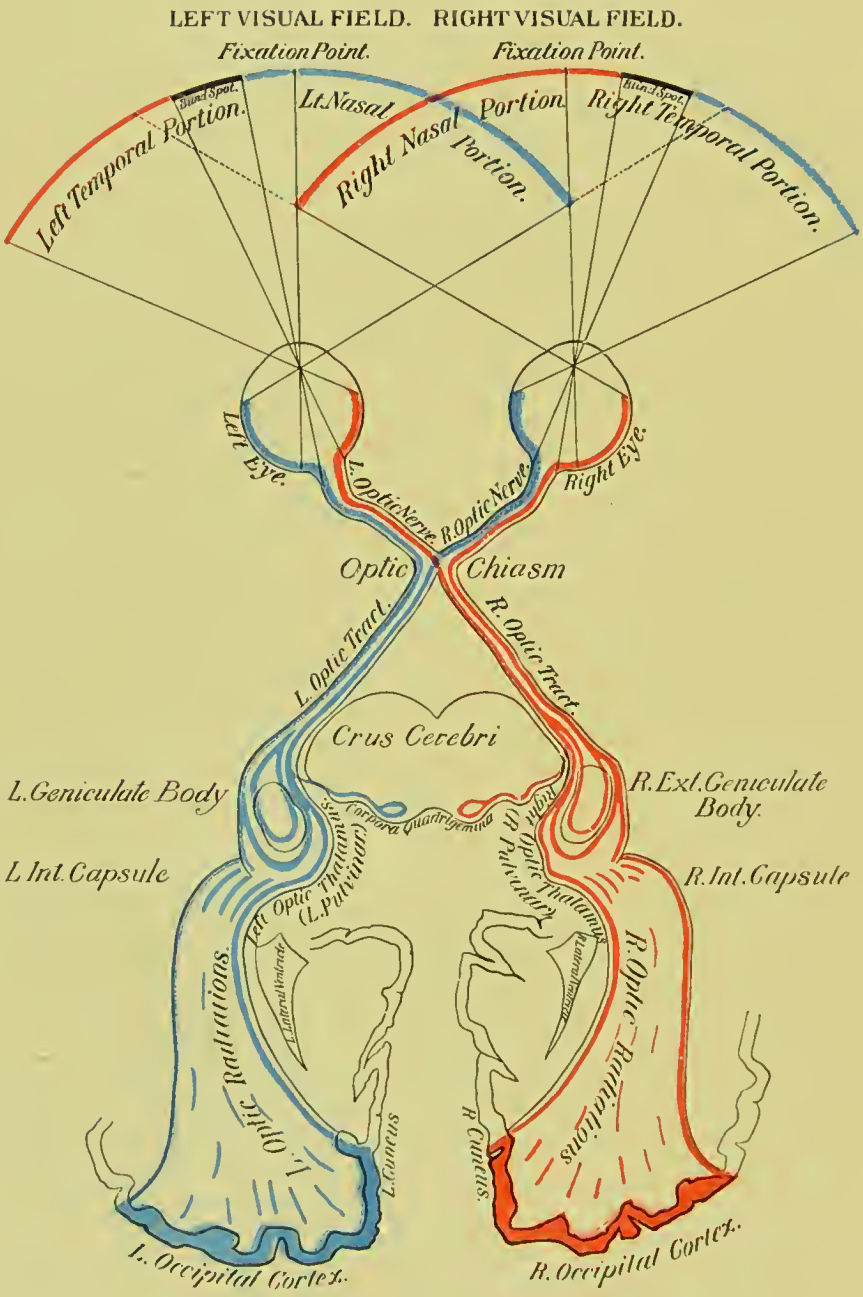
We are now in a position to discuss intelligently the data for the localization of intra-cranial affections which are furnished by lesions involving the path of the optic nerve fibres posterior to the orbit.

Such binocular subjective symptoms may be conveniently classified under two heads, viz.:

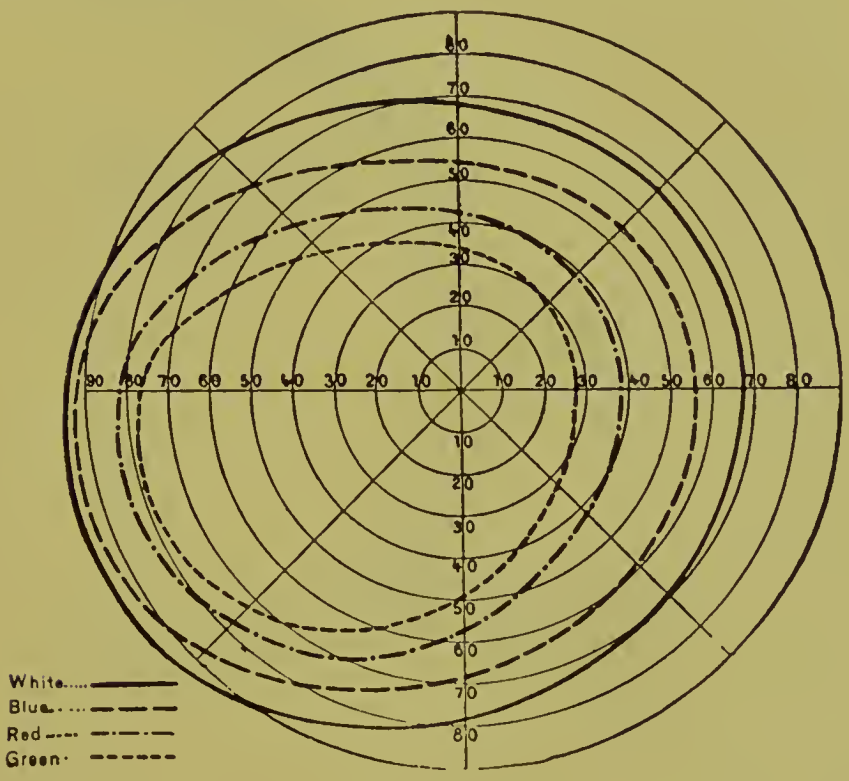
1. Changes in the form and extent of the visual fields constituting the various forms of hemianopsia.

2. Amaurosis and amblyopia (or, more correctly, cortical blindness), and the varied forms of psychic visual disorders.

A knowledge of the normal boundaries of the visual fields for form and color is essential for a recognition of pathological defects in its dimensions or outline. When the eyes are fixed upon a given object its retinal image is formed upon the macula lutea of each eye, the most sensitive portion of the retina, and at the same time rays of light emanating from objects on all sides of the object of fixation enter the pupil, and form more or less distinct images upon peripheral parts of the retina. It is evident that the farther such visual images are projected into space, the larger the resulting visual field will be, but it will always represent a certain number of degrees of the circumference of a circle, however large or small such a circle may be. It is evident also that the vision will be more extensive on the temporal side than in other directions, because the outlines of the orbit and the bridge of the nose will obstruct the course of peripheral rays in other directions, and that the limits in individual cases will vary slightly according to the conformation of the face. The physiological limits of the visual field for form are, approximately, 90° outward, 60° above, 50° on the nasal side, and 65° below. The field


LEFT VISUAL FIELD.
RIGHT VISUAL FIELD.
Fixation Point.
Fixation Point.
Blind Spot.
Blind Spot.
Left Temporal Portion.
Lt. Nasal Portion.
Right Nasal Portion.
Right Temporal Portion.
Left Eye.
L. Optic Nerve.
R. Optic Nerve.
Right Eye.
Optic Chiasm
L. Optic Tract.
R. Optic Tract.
Crus Cerebri
L. Geniculate Body
R. Ext. Geniculate Body.
L. Int. Capsule
R. Int. Capsule
Left Optic Thalamus (L. Pulvinar)
Corpora Quadrigemina
Right Optic Thalamus (R. Pulvinar)
L. Optic Radiations.
R. Optic Radiations.
L. Occipital Cortex.
R. Occipital Cortex.

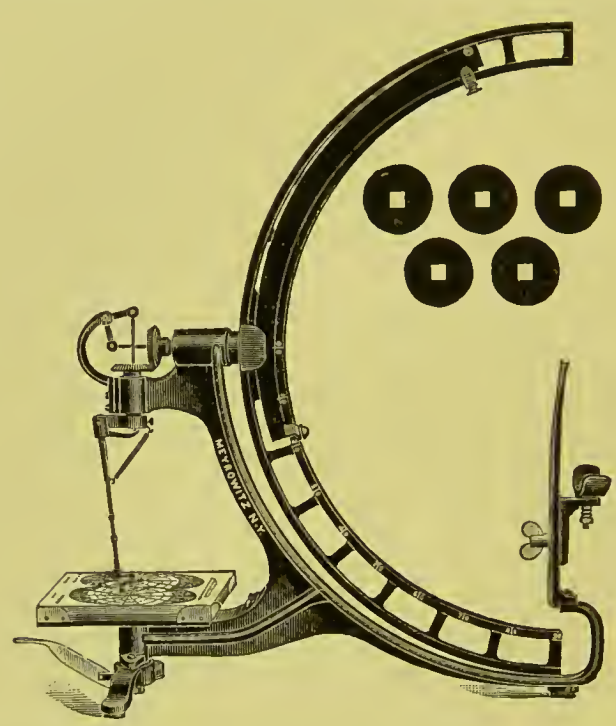
for color is not coextensive with that for form, and differs with the different colors, that for blue being the largest, and that for green the smallest. The accompanying chart illustrates the physiological limits of the visual fields.



The examination of the visual field is best made by means of the perimeter. There are various patterns of this instrument in the market. The latest and best design is made by E. B. Meyrowitz, of new York, a cut of which is appended. The special feature of this instrument is that it is self-recording, a desideratum which

has long been sought but never practically attained before.

A perimeter will hardly be found in the office of the general practitioner, and, in its absence, an approxi-



mate estimate of any pathological change in the visual field can be gained in the following way:

The patient, seated directly in front of the physician and about two feet distant, fixes with his left eye, for instance, the right eye of the examiner, or *vice versa*, the other eye of both physician and patient being closed. Then the farthest point in all directions, at

which the movement of the physician's hand (held midway between their two faces,) can be seen by the patient while the position of his eye is unchanged, gives the outline of his visual field. By comparing this with the physician's field, simultaneously delineated, any decided abnormality is detected.

HEMIANOPSIA.

By this term is understood a complete or partial loss of sight, affecting one-half of each visual field. Care is to be exercised not to confound it with blindness of one eye.

Hemianopsia may be classified as heteronymous or homonymous. In the former, unsymmetrical parts of the visual field are involved, and it may again be designated as heteronymous medial hemianopsia when the nasal half of each field is defective, or heteronymous temporal hemianopsia when the loss of vision is confined to the temporal half of each retina. The dividing line between the seeing and the non-seeing areas may or may not pass through the fixation point, and the blindness may be partial or absolute—that is, the hemianopsia may be complete or incomplete. Since the fields of vision are projected from the opposite sides of the retina, it is evident that medial hemianopsia signifies blindness of the temporal half of each retina, and temporal hemianopsia, blindness of the nasal half. Either variety of heteronymous hemianopsia indicates

an affection of the chiasm, and is strongly suggestive of a syphilitic gumma, for gummous meningitis at the base of the brain is, next to endarteritis, the most common form of brain syphilis, and the most frequent situation of the affection is in the immediate vicinity of the chiasm. Temporal hemianopsia, as a result of syphilis, occurs, it is estimated, twice as frequently as homonymous hemianopsia from the same cause.

In medial hemianopsia the uncrossed bundles of fibres of each tract are affected, and therefore the lesion is to be located at the outer edge of the chiasm on either side. In temporal hemianopsia the lesion involves the crossed bundles of each tract, and it can only result from a morbid process affecting the central part of the chiasm from before backward, or at its anterior or posterior angle. Accumulation of fluid in the third ventricle with hydrops of the infundibulum would exert transverse pressure on the chiasm, with such visual manifestations.

Homonymous hemianopsia, that is, a loss, more or less complete, of functionally associated and corresponding portions of each visual field, right or left, can only be explained by a lesion affecting the fibres of one optic tract, and this may be situated at any point between the chiasm and the cortex. The designation of right or left homonymous hemianopsia, it will be understood, refers to field and not to retina. The presence or absence of the light reflex and of visual hallu-

cinations (photopsia, etc.) is of importance in more exactly locating the lesion. The optic tract may be compressed by the exudation attending basilar meningitis, by a syphilitic gumma (the most frequent cause) or other neoplasm, or by a hemorrhage. It is evident that since visual impressions are interrupted in the affected tract, sensory reflex iridoplegia will necessarily result. There will be no contraction of the pupils when light is thrown upon the blind halves of the retinae, but the light reflex will be preserved when the light is thrown on the seeing part of either retina, transmission being uninterrupted along the uninjured tract.

The same condition, viz., "hemianopic pupillary inaction" is present in homonymous hemianopsia due to a lesion of the pulvinar, and in the absence of involvement of the nerves supplying the ocular muscles, and other phenomena which would almost of necessity accompany a lesion of one tract, this symptom warrants a diagnosis of a lesion at this point. When hemianopic visual disorders are caused by disease in the course of the optic radiation, or in the cortical visual area, the pupillary light reaction is preserved, and this is true whether the blind or the seeing portion of the retina is illuminated, and irrespective of any conscious sensation of light. Homonymous hemianopsia may occur suddenly as the sole manifestation of a cerebral hemorrhage. If, under such circumstances, the pupillary light reflex is preserved, the presence or absence of visual hallucina-

tions may enable one to decide whether the hemorrhage is cortical, or situated in the path of the optic radiation. If the visual area in the occipital cortex is destroyed, there can be no photopsia or visual hallucinations. Hence, if they exist in the affected half of the visual field the lesion is not cortical, but when they are wanting, we possess no data for exact localization in one or other of these circumscribed areas.

Vialet, of Paris, in "*Annales d'Oculistique,*" Paris, April, 1894, thus defines the boundaries of the cortical visual area. It "occupies the entire extent of the internal surface of the occipital lobe, embracing the cuneus and the fusiform lobes, being bounded anteriorly by the internal perpendicular fissure (parieto-occipital?), above by the superior edge of the hemisphere, below by the inferior edge of the third occipital convolution, and behind by the occipital lobe."

Different portions of this area are in relation with distinct parts of the field of the opposite eye. "The macular region corresponds to the cuneus and, perhaps, to the first occipital convolution. In the remainder of the occipital cortex known as 'Nothnagel's memory centre for visual impressions,' the anterior part corresponds to the inferior part of the opposite field, the lateral part to the outer, the posterior parts to the upper portions of the field. Hence in partial destruction of the occipital cortex, the visual disturbance varies

greatly according to the location of the diseased focus." (Knies).

There seems to be a separate centre for the perception of color, for hemianopsia for color sometimes exists without other defect. The apex of the occipital lobe is the spot where a lesion produces most complete and pronounced hemianopsia, but injuries to the outer and medial surfaces also produce this defect to a lesser degree, manifested as "hemianopic peripheral scotomata with practically no subjective sensations, and not detected with the perimeter without great care." It should be borne in mind that cortical lesions are always homonymous, and this feature will aid in a differential diagnosis between such disorders and scotomata and contractions of the fields due to affections of the retina and optic nerve, which occur in the course of cerebral tumors, dementia paralytica, tabes, etc.

Defects of the visual field without ophthalmoscopic findings also occur in hysteria and uraemia, and the reader is referred to the chapter on reflex neuroses for a discussion of such disorders.

CROSSED AMBLYOPIA.

It would seem from the occurrence of crossed amblyopia, that is, dimness of sight in the opposite eye, generally with concentric diminution of the field, and with some restriction of the field on the same side, that there is a higher visual centre where impressions from

both fields are united. There have been a few recorded autopsies of cases presenting crossed amblyopia during life, in which the posterior and inferior part of the parietal lobe known as the "angular gyrus" was diseased. Thus pathological evidence would seem to point to this locality as the seat of a higher visual centre, and Ferrier has demonstrated the existence of such a centre at this spot in animals. The theory of crossed amblyopia was first advanced by Charcot, and it is supported by Gowers but discredited by others.

There are various forms of transient amblyopia which can only be explained by temporary disorders of the circulation in the visual centres. Such transient amblyopia may be manifested as hemianopic defects, symmetrical scotomata, central or peripheral, or as more or less complete blindness associated or not with scintillation, or the appearance of sparks or flashes of light. Such attacks appear more frequently in females, and in persons of neurotic temperament. They are explained by a vaso-motor disturbance in the visual areas, and it is important to understand that they are not, as a rule, of serious import. Occasionally they are the precursors of an epileptic attack. More frequently they are associated with cephalalgia and vertigo.

AMAUROSIS AND AMBLYOPIA. PSYCHIC VISUAL DISORDERS.

Sudden blindness of both eyes may result from a simultaneous affection of both visual areas. Monocular

blindness points to an affection of the optic nerve in front of the chiasm, or to an intra-ocular affection. Cortical disorders, it will be remembered, are always binocular, and are attended with preserved pupillary reaction. Meningitis of the convexity of the brain with exudation may occasion such loss of vision. It may result from extreme loss of blood, producing anaemia and loss of sensibility of the visual centres. In such cases the vision will be recovered with the returning circulation. It may be due to malaria. It may be of toxaemic origin, especially in uraemia. It may be due to reflex irritation or be a manifestation of hysteria. The visual disorders associated with these affections are discussed in the chapter on reflex neuroses.

The opposite condition, viz., undue excitability of the visual centres manifested by photophobia and hyperaesthesia of the retina, phosphenes, etc., unaccompanied with inflammatory conditions of the eye may be a part of a general nervous excitability, or may be an early symptom of meningitis or encephalitis.

There will be no difficulty in diagnosing a case of sudden blindness due to uraemic intoxication, for it is always associated with other symptoms, such as headache, vomiting, stupor and convulsions. It is caused by an affection of the visual centres either from a toxic element in the blood, or, perhaps, as has been suggested, by an anaemia consecutive to an extensive effusion into the cerebral ventricles. The exact pathology

is undetermined. The pupillary reaction to light is ordinarily preserved, and unless complicated with a previous albuminuric retinitis, there are no ophthalmoscopic changes, and the sight returns with the subsidence of the other uraemic manifestations. Uraemic amaurosis is said to occur most frequently with post-scarlatinal nephritis, and next with the contracted kidney, and it is sometimes met with in the nephritis of pregnancy. Similar visual disturbances occasionally appear in the later stages of diabetes.

It remains to discuss certain psychic visual disorders.

HALLUCINATIONS OF SIGHT, that is, "false interpretations and judgments of real sensorial phenomena," are an indication of mental disorders. The pupils dilate or contract as the fancied object apparently recedes or approaches. This circumstance may be of use in the treatment of delirium or insanity. Such symptoms are also sometimes of toxic origin. See chapter on toxic amblyopia.

A peculiar and interesting form of visual defect styled by Fuchs "soul blindness," sometimes results from a diseased focus situated in the vicinity of the localities implicated in cases of hemianopsia and crossed amblyopia, but distinctly separated therefrom. The patient sees, but he has lost all memory of visual impressions, and, therefore, objects are unrecognized. He cannot read because he has lost all memory of the meaning of

the characters (alexia). Or he may recognize objects, but cannot recall their names, a condition constituting word blindness.

Soul blindness is usually a transient symptom associated with disease of another portion of the brain. Knies says that it is very rare as an isolated symptom, that it occurs quite often with progressive paralysis of the insane, but is always temporary.

An interruption in the course of the commissural fibres uniting the visual areas with the higher intellectual centres explains another interesting class of psychic visual disorders which may be briefly mentioned, viz. :

Motor Alexia.—This is very similar to soul blindness and word blindness, but differs from those conditions in that the names of objects seen cannot be spoken, although remembered.

Dyslexia.—"He can read aloud, but after uttering a few words, a peculiar uncomfortable feeling is experienced which compels him to stop. This condition is temporary, but often followed by severe cerebral symptoms."

Paralexia.—A condition in which "single letters, syllables or words are omitted in reading or confused with others having a similar sound."

Agraphia.—"In which he cannot copy, although the movements of the hand and arm are unimpaired and he can write from dictation."

PARAGRAPHIA.—In which there is a "confusion or omission of letters, syllables and words in writing."

Such conditions as the above are usually transitory. The precise location of lesions causing them cannot be definitely determined, but it may be stated that if the motor or visual symptoms are the more pronounced, the disease is located nearer the motor or the visual centres respectively.

CHAPTER VI.

A TABULATED STATEMENT OF DISEASES WITH MORE OR LESS CHARACTERISTIC EYE SYMPTOMS.

ABDOMINAL GROWTHS.

More or less pigmentation of the skin of the eyelids.

ADDISON'S DISEASE OF THE SUPRA RENAL CAPSULES.

Pigmentation of the skin of the lids and of the sclera.

ALBUMINURIA.

Retinitis, and neuro-retinitis.

ALCOHOLISM.

Paretic mydriasis. Paralysis of accommodation, or spastic myosis in the early excitable stage. Paralysis of the external ocular muscles. Ptosis.

ANAEMIA (CEREBRAL).

Paretic mydriasis

ANAEMIA (CONSTITUTIONAL).

Paralysis of accommodation, choroiditis, retinitis and retinal hemorrhages.

ANEURISM OF ORBITAL ARTERY, OR INTERNAL CAROTID.

Exophthalmus.

AORTA AND ARTERIA INNOMINATA, ANEURISM OF.

Reflex spastic mydriasis on the side of the lesion. Retinal pulsation.

APOPLEXY.

Dilatation or contraction of the pupils distinguishes it from embolism, in which the pupils are unaffected. Spastic myosis in premonitory stage. When, during a seizure, mydriasis occurs after a previous myosis it is an unfavorable symptom, signifying increasing pressure. Nystagmus. Homonymous hemianopsia.

APOPLEXY OF CORTEX OR CORONA RADIATA.

Eyes and extremities paralyzed on the same side. Eyes deviate toward the side of the lesion.

APOPLEXY OF THE CRUS OR PONS VAROLII.

Eyes and extremities paralyzed on opposite sides. Eyes deviate away from the side on which the lesion exists.

APOPLEXY OF THE PONS.

Spastic myosis.

APOPLEXY OF VENTRICLES.

Spastic myosis.

ATHEROMA.

Conjunctival hemorrhage. Intra-ocular hemorrhages.

ATROPHY, PROGRESSIVE, MUSCULAR.

Ocular paralyses.

BASEDOW'S DISEASE OR EXOPHTHALMIC GOITRE.

Diminished frequency of winking. Spasm of the levator of the upper lid. (Abadie's sign). Widening of the palpebral fissure, owing to contraction of Mueller's muscle. (Stellwag's or Dalrymple's

sign). Loss of associated movement of the upper lid and the eye-ball. (Von Graefe's sign). Ex-ophthalmus.

BRAIN, ABSCESS OF.

Neuritis.

" **BASILAR AFFECTIONS OF.**

Loss of pupillary reflexes. Homonymous hemianopsia. Paralyses of ocular muscles.

" **CEREBRAL CORTICAL AFFECTIONS.**

Conjugate ocular paralyses, (or ophthalmoplegias.) Loss of voluntary movements of the eyes, with preservation of involuntary or reflex movements of pupil and eye-ball. Eyes paralyzed on side opposite the cerebral lesion. Eyes deviate towards side of lesion. Psychic visual disorders.

" **CEREBRAL AFFECTIONS WITH INCREASED INTRA-CRANIAL PRESSURE.**

Paretic mydriasis ordinarily. Sometimes reflex spastic mydriasis.

" **CEREBELLUM, AFFECTIONS OF.**

Nystagmus.

" **CONCUSSION OF.**

Sluggish action of pupils without marked dilatation or contraction.

" **HYPERAEMIA OF.**

Spastic myosis.

" **TUMOR OF.**

Nystagmus. Paretic mydriasis. *Choked disc. Choked neuritis.* Atrophy of optic nerve.

Homonymous hemianopsia, when pressure is exerted upon fibres of one tract.

CHOLERA.

Conjunctival hemorrhage. Anesthesia of cornea. Neuro-paralytic keratitis. Loss of the light reflex indicates a fatal termination even in apparently mild cases. Preservation of the light reflex warrants a favorable prognosis, even in severe cases. Black patches appear in the sclerotic below the cornea in severe cases. They are of irregular form and size and tend to coalesce. Their presence is of very unfavorable significance.

COMA, ALCOHOLIC OR URAEMIC.

Mydriasis.

COMA, SYPHILITIC.

Myosis and reflex iridoplegia.

DEATH, SIGNS OF.

Opacity and insensibility of the cornea. Desiccation of the sclera. Abolition of pupillary reflexes. Absence of the red reflex from the fundus.

DENTAL AFFECTIONS.

Various forms of inflammation of the cornea. Nictitation.

DIABETES.

Eczema of the eyelids. Conjunctival hemorrhage. Ulceration of the cornea. Paralysis of the external ocular muscles and of the accommodation. Cortical cataract. Retinitis and neuro-retinitis.

Atrophy of the optic nerve. Degeneration of the retinal vessels and hemorrhages. Lagrange found, in 52 cases of diabetes, 13 of intra-ocular hemorrhage and the same number of cases of cataract. (See *"Arch. d'Ophth.,"* Jan., 1887). Galezowski found in 144 cases of diabetes, 5 of paresis of accommodation, 4 of keratitis, 7 of iritis, 4 of glaucoma, 46 of cataract, 27 of retinitis, 31 of amblyopia, 3 of amotio retinae, and 3 of atrophy of the optic nerve. (See *"Jahr. f. Aug.,"* 1883, p. 297).

DIGESTION, DISORDERS OF.

Styes. Nictitation.

DIPHTHERIA.

Diphtheritic conjunctivitis. Paralysis of the external eye muscles rare; of accommodation more frequent.

EMBOLISM, CEREBRAL.

No pupillary symptoms; in contradistinction from apoplexy.

EPILEPSY.

Paretic mydriasis during the seizure or spastic myosis. Hippus as consciousness returns and frequently during the intervals. Spasms of the ocular muscles.

FEVER, PUERPERAL AND TYPHOID.

Metastatic suppurative choroiditis.

FEVER, RELAPSING.

Iritis.

FIFTH NERVE, AFFECTIONS OF.

Reflex spastic myosis.

FOURTH VENTRICLE, LESIONS IN.

Nuclear ocular paralyses affecting separate nuclei of the third nerve, or successive implication of its various branches. Also total paralysis of all the muscles of both eyes.

FRIEDREICH'S DISEASE (HEREDITARY ATAXIA).

Nystagmus.

GOUT.

Retinitis.

HEART, AORTIC INSUFFICIENCY.

Alternate reddening and pallor of the optic disc.

HEART, ENDOCARDITIS.

Embolism of the arteria centralis retinae.

HEART, ORGANIC AFFECTIONS OF.

Oedema of lids. Venous hyperaemia of retina and pulsation of retinal arteries. Seen with valvular affections, fatty heart and aortic insufficiency.

HEART, HYPERTROPHY OF LEFT VENTRICLE.

Retinal hemorrhages.

HEART, VALVULAR LESIONS OF.

Retinal hemorrhages.

HELMINTHIASIS.

Reflex spastic mydriasis.

HEPATIC AFFECTIONS.

Pigmentation of the skin of the lids. Coloration of sclera.

HYDRAEMIA.

Oedema of lids.

HYDROCEPHALUS.

Paretic mydriasis. Neuritis and atrophy of optic nerve.

HYSTERIA.

Chromidrosis. Epiphora. Ptosis. Spastic myosis (during a hysterical convulsion). Hippus. Hyperaesthesia of the retina. Spasm of accommodation. Amblyopia. Contraction of the visual field. Sudden onset, erratic course, sudden disappearance.

INSANITY.

Monocular mydriasis and paralysis of accommodation are suspicious premonitory signs, as is also transient recurrent mydriasis.

KIDNEY, DISEASES OF.

Degeneration of the retinal vessels with or without hemorrhages. Retinitis and neuro-retinitis. Amblyopia. Oedema of lids.

LEPROSY.

Leprous nodules in eyelids, conjunctiva, cornea and iris. Anaesthetic spots and white patches in the lids. According to Lopez (*"Archiv. f. Aug.,"* XXII, 2 and 3) "the eye is affected in half the cases, the eye with its appendages in all cases." Knies.

LUNG, DISEASE OF APEX.

Reflex spastic mydriasis.

MALARIA.

Chronic superficial non-suppurative keratitis. Sensitiveness of supra-orbital nerves. Retinal hemorrhages. Choroiditis.

MANIA.

Reflex spastic mydriasis.

MASTURBATION.

Paralysis of accommodation. Hyperaesthesia of the retina.

MELANCHOLIA.

Reflex spastic mydriasis.

MENINGITIS, CEREBRAL.

" **ACUTE.**

Mydriasis or myosis. Photophobia. Injection of conjunctiva.

" **CHRONIC.**

Interstitial and peri-neuritis. Atrophy of the optic nerve.

" **OF THE CONVEXITY.**

Cortical blindness or hemianopsia with preserved pupillary light reflex. Hyperaesthesia of the retina. Photophobia, phosphenes, etc.

" **CEREBRO-SPINAL.**

Eye symptoms frequent. Conjunctivitis in early stages. Later oedema of conjunctiva, denoting exudation in cranial cavity. Strabismus. Nystagmus. Spastic myosis in early stages. Reflex spastic mydriasis from pinching the skin at the back of the neck.

(Parrot's sign). Hippus. Choroiditis. Photophobia. Neuritis.

MENINGITIS, SPINAL.

Spastic mydriasis in the early stage.

" TUBERCULAR.

Strabismus. Nystagmus. Ocular paralyses. Spastic myosis in early stage. Rapid alternation of myosis and mydriasis. Paretic mydriasis in later stages in contra-distinction from cerebro-spinal meningitis, in which it is rare. Tuberculosis of the choroid. Homonymous hemianopsia.

MENINGEAL HEMORRHAGE.

Nystagmus. Hemianopsia.

MENSTRUATION, DISORDERS OF.

Styes.

MYELITIS, ACUTE AND CHRONIC.

Neuritis or simple atrophy of the optic nerve.

MYXOEDEMA.

Thickening and swelling of the lids.

NEPHRITIS.

See diseases of the kidney.

NEURALGIA OF THE FIFTH NERVE.

Paralysis of accommodation.

NEURITIS, MULTIPLE OR PSEUDO-TABES.

Axial neuritis with central scotoma. Absence of pupillary symptoms, in contra-distinction from true tabes.

NICOTINE POISONING.

Spastic myosis. Retro-bulbar neuritis with central scotoma.

PARALYSIS.

" AGITANS.

Tremor of the lids. Ptosis.

" GENERAL (PARALYSIS OF INSANE. PARESIS).

Monocular mydriasis and paralysis of the accommodation and transient recurrent mydriasis are suspicious premonitory symptoms. Paretic mydriasis is an early symptom. The "Argyll-Robertson pupil" is found in fifty per cent. of the cases. Anisocorea. Paretic myosis. Optic neuritis. Atrophy of optic nerve. Soul blindness. Sudden development and transient duration of ocular symptoms, similar to multiple sclerosis and tabes.

POLIO ENCEPHALITIS SUPERIOR (INFLAMMATION OF THE FLOOR OF THE FOURTH VENTRICLE).

Progressive paralysis of the ocular muscles is the essential feature.

PONS VAROLII, LESIONS OF.

Nystagmus. Associate ocular paralyses in horizontal lines. Spastic myosis. An isolated lesion of one side produces paralysis of the external rectus on the same side.

PYAEMIA.

Metastatic suppurative choroiditis. Retinal hemorrhages.

RACHITIS.

Cortical or laminated cataract.

RHEUMATISM.

Paralysis of external ocular muscles, usually of one eye, and affecting one or more contiguous branches of the nerve, such as the superior rectus and levator palpebrae superioris. Iritis with gelatinous exudation.

SCLEROSIS, MULTIPLE.

Nystagmus, a frequent and valuable diagnostic sign. Ocular paralyses characterized by sudden development, transient duration and variable course, similar to syphilis and tabes. Hippus. Paralysis of accommodation. Impairment of vision, but rarely complete blindness. Central scotoma. Irregular or concentric contraction of the visual field. Neuritis.

SCROFULA.

Eczema of lids. Styes. Ciliary blepharitis. Conjunctivitis. Pustules and abscesses of the cornea. Phlyctenular conjunctivitis and keratitis. Choroiditis.

SNAKE POISONING.

Retinal hemorrhages.

SKIN, EXTENSIVE BURNS OF.

Reflex spastic mydriasis (skin reflex).

SPINAL CORD.

" INFLAMMATION AND CONGESTION OF. SPINAL IRRITATION.

Spastic mydriasis occurs in the early stages.

SPINAL CORD, DEGENERATIVE DISEASE OF.

Nystagmus.

SYPHILIS.

Every tissue of the eye, except the lens, is affected. Inflammations of the lids, orbit and lychrymal passages. Arrest of development, such as microphthalmus, etc., in the congenital form. Periostitis and caries of the orbit. Paralytic affections of the lids and external ocular muscles. Various muscles are suddenly, successively and transiently involved. Parenchymatous keratitis. Inflammation of the sclera with gummata. Mydriasis with loss of accommodation. Gummous iritis. Choroiditis. Degeneration of the retinal vessels with or without hemorrhages. Retinitis and neuro-retinitis. Atrophy of the optic nerve. Heteronymous and homonymous hemianopsia. Zimmerman, of Milwaukee, says, in "Knapp's Archives," Jan., 1895, that only about 15 per cent. of the cases of brain syphilis are without ocular symptoms.

TABES.

Anaesthesia of the skin of the lids of the conjunctiva and cornea, with false localization of sensation. Paresis of orbicularis palpebrarum. Narrowing of the palpebral fissure. Ptosis. Paralysis of the ocular muscles, sudden in development and transient in duration, similar to syphi-

letic paralyses and to those which occur in multiple sclerosis.

Spastic mydriasis may be a premonitory symptom. Paretic myosis occurs in 23 per cent. of the cases. The "Argyll-Robertson pupil" is a very characteristic symptom and occurs in 70 per cent. of the cases. In 25 per cent. it is an early symptom. Reflex iridoplegia, or failure of all the pupillary reflexes. The reaction to light fails first, followed by loss of reaction with accommodation and convergence, and lastly the skin reflex is lost.

Anisocorea occurs in 34 per cent. of the cases. Reflex iridoplegia is a valuable diagnostic point between true and false tabes, or multiple neuritis. In the latter, myosis and reflex iridoplegia are wanting. Atrophy of the optic nerve.

Ocular symptoms may appear very early, even many years before the ataxic symptoms. Gowers relates a case where twenty years elapsed between blindness optic nerve atrophy, etc., and the onset of ataxia.

When spinal symptoms are well marked ocular symptoms are often latent or absent, and the reverse is also true, viz., when ocular symptoms are marked the spinal symptoms are slight or absent and may be long delayed.

TRICHINOSIS.

Oedema of the lids and paralysis of accommodation.

TUBERCULOSIS.

Periostitis and caries of the orbit. Tubercular nodules in eyelids, conjunctiva, iris and choroid. Iritis with grayish-red nodules.

TYPHOID FEVER.

Anaesthesia of the cornea, neuro-paralytic keratitis. Hippus in stage of cerebral manifestations. Metastatic suppurative choroiditis. Intra-ocular hemorrhages.

URAEMIA.

Mydriasis is a premonitory sign. Sudden failure of vision.

UTERINE AFFECTIONS.

Pigmentation of the skin of the lids. Paralysis of accommodation.

URTICARIA.

Reflex spastic mydriasis.

PART SECOND.

REFLEX NEUROSES.

CHAPTER VII.

THE RELATION OF OCULAR AFFECTIONS TO FUNCTIONAL NERVOUS DISEASES.

The term neurosis may be used synonymously with a functional nervous affection. By either expression we understand a disorder of the nerves, or nerve centres, of a purely functional nature, and unassociated with known organic structural changes. It is not asserted that such changes may not exist, but that they have not been recognized. A functional nervous disease may originate in some irritation or lesion in a part distant from that in which the symptoms are manifested. In such a case the disease is designated as a reflex neurosis, and the reflex influence of affections of the eye in causing functional nervous diseases is the theme of the present chapter. The converse of this, viz.: the discussion of reflex neuroses, as they are manifested by functional eye diseases dependent upon distant foci of irritations, will be reserved for subsequent consideration.

Functional nervous diseases may be conveniently classified as general and local. In the former class belong epilepsy, chorea, neurasthenia and hysteria. Very much has been written during the past few years upon the relation of eye-strain to such affections, and the ten-

dency of specialism in medicine to beget narrowness of view, and of enthusiastic study in one direction to distort the judgment is nowhere more clearly exemplified. Mere coincidence has been many times mistaken for cause and effect. The origin of almost all nervous affections has been assigned by some over-zealous ophthalmologists to ocular disorders, and the exaggerated importance thus given them has been denied by others, some of whom undoubtedly underrate their influence in such directions. The truth, as I shall endeavor to show, lies between these two extreme views.

The study of neuroses is one of great importance, and whatever promises to extend our knowledge of their causes, and thus to teach us how to prevent their development, or on the other hand, offers additional means of cure is of direct value and merits careful consideration. The recorded observation and experience of multitude of skilled and trustworthy physicians seem to demonstrate that ocular affections are both a cause and an effect of functional nervous diseases.

The wear and tear of modern life consequent upon the competition of business, the feverish excitement and anxiety of speculation, the demands of social and fashionable life with its mental and physical exhaustion, the excitement of modern fiction and the drama, are some of the universally recognized exciting causes of such diseases. Thus an inherited neurotic temperament transmitted from parent to child is a fre-

quent predisposing cause, and the extreme and increasing prevalence of nervous disorders is thus readily understood. When a person of this neurotic temperament, hereditary or acquired, is subjected to any special source of nervous exhaustion the effect of such strain is intensified, and is followed, in many instances, by pronounced and far-reaching effects. The fact that such overwork, anxiety, loss of sleep, and so forth is often well borne without obvious injury by persons in robust health is no proof of the incorrectness of this statement. Similarly, any organ, although somewhat crippled by reason of structural weakness or difficult or painful exercise of function, may do its work without apparent detriment when the body as a whole is in a condition of perfect health, but when the vital powers are enfeebled from any cause, such as lack of nutrition, overwork bodily or mental, or from actual disease, the weak organ is the first to suffer and the slowest to recover.

We recognize in certain functional eye-diseases both the cause and the effect of general neuroses such as those enumerated. The faculty of vision is a most complex one, demanding the exercise of various and intricate nervous activities. The first requisite for binocular single vision is that an image of the object shall be formed upon corresponding portions of each retina. Otherwise, the two retinal impressions cannot be blended into a harmonious single perception, and diplopia results. In order that the images may fall upon cor-

responding retinal points, the functional integrity of the four recti and the two oblique muscles, by which the harmonious associated movements of the two eyes are obtained, is essential. This means uninterrupted and harmonious innervation along the lines of the third, fourth and sixth nerves, proceeding from physiologically intact centres, which preside over and regulate the ocular movements. The normal exercise of the faculty of accommodation is also essential to perfect vision, as well as the movements of the iris, calling for exercise of other fibres of the third and of the sympathetic nerves. Supposing these factors to be physiologically and anatomically perfect, and the dioptric media to be transparent and of proper refractive power, the conduction of visual impressions through the optic nerve must be unimpaired, and the visual centres must be in healthy condition, in order that the impressions thus received may be converted into intelligent perceptions of form, size and color, and that accurate conceptions of the nature, distance and position of objects may be obtained. When we remember that during all our waking hours the eyes are in constant use in both distant and near vision, and when we consider the close application required of the student, the artist, the professional man, the book-keeper and the skilled mechanic, we are in a position to realize the amount of nervous energy which is thus called for, and to appreciate the strain resulting from any abnormality of structure or function

in the visual apparatus, and the influence which such a strain exerts in the development and maintainance of functional neuroses in persons of neurotic temperament. In all such cases, it should be remembered that eye-strain is an important factor to be considered.

OCULAR AFFECTIONS AS A CAUSATIVE INFLUENCE IN EPILEPSY.

The literature of the past few years contains frequent references to the subject, and many cases have been reported in which cures have followed correction of refractive or muscular anomalies of the eyes. Doubtless errors in diagnosis have sometimes been made, and in other instances sufficient proof of recovery has not been offered; nevertheless the testimony of so many investigators, many of them men of recognized ability and trustworthiness, is deserving of careful consideration. Leaving out of the question those cases of epilepsy consecutive to injuries of the skull, or associated with organic brain disease, there remain many others of so-called "idiopathic epilepsy" in which no pathological changes have been discovered. These cases occur in persons of a strongly neurotic temperament, with whom there is often a family history of previous epilepsy or insanity. Inherited syphilis often exists in these individuals with defective development of the brain. The latter however may exist without syphilis, and be the result of other influences. The remote cause, viz., the constitu-

tional predisposition thus induced, is of vastly more importance than the exciting cause. The latter is often apparently insignificant and frequently overlooked. It may be dental irritation, helminthiasis or other intestinal disorder, or a reflex influence from sexual or other nervous excitation. Frequently it is some strong mental impression, such as fright, excitement or sudden grief. These conditions *alone* are incapable of originating true epilepsy, but they are all recognized as adequate exciting causes of the disease when *added* to a pronounced predisposition to epilepsy. Any one of them may on an appropriate occasion serve as a match to fire an explosion of nervous energy resulting in a convulsive seizure. A repetition may occur from the same or a similar cause, and the susceptibility of the individual increases with each attack until they recur without assignable cause, and thus true idiopathetic epilepsy is developed. A single convulsive seizure directly due to a reflex irritation, as in children during dentition, or with acute indigestion, may strongly resemble true epilepsy, but in the absence of repetition could hardly be so diagnosed. If a transient condition such as those mentioned, or a violent fit of anger or fear may thus become the exciting cause of convulsive attacks which continue to recur without the repetition of the initial cause, it seems much more plausible to assign a causative influence to a *permanently* active source of nerve waste such as is found in eye-strain, and it is a signifi-

cant fact that Semeling ("*Charité Annal.*," XI, p. 389) asserts that anomalies of the eye occur in 20 per cent. of all epileptics.

Another argument in support of the influence of eye-strain is afforded by the fact that visual aurae precede an attack in a large number of cases of epilepsy. Gowers says, ("Diseases of the Nervous System," Vol. 2, p. 739): "A visual warning is twice as frequent as all the other special sense aurae together. It may be a sudden loss of sight, but is more frequently a visual sensation, a flash of light or sparks, or flashes of color. Usually many colors are seen, red and blue most frequently. It may be an elaborate sensation, a vague beautiful vision, or a definite image of some object, for instance, an old woman with a dress of some certain color, ugly faces, animals, etc."

An aura, either sensory or motor, "gives us information of the functional region of the brain where the process of the fit begins," and such visual aurae as enumerated clearly indicate that in many cases the visual centre is the locality where the "discharge" commences. Aural warnings associated with the sense of vision, or of varied nature and location are more common in epilepsy associated with organic cerebral lesions than in the idiopathic variety, and they do not usually indicate an ascending irritation from the part where the peculiar sensation is felt, but are rather the expression of a central irritation referred to the extremity first affected.

Clinical experience teaches that the converse may be true viz., that peripheral ascending irritation is sometimes the cause of the attack. Gowers mentions a case which illustrates this fact. A boy received a severe cut on one of the fingers of the right hand which was followed by epilepsy. The spasms always commenced in the hand and were arrested by amputation of the injured finger.

Visual aurae may be indirectly dependent upon eye-strain, and the cure of epilepsy by correction of ocular anomalies is explained, as in the above case, by the removal of the cause of peripheral irritation. The path by which such influence travels from the eye to the brain is uncertain, but the hypothesis that anaemia of the brain, due to vaso-motor spasm, is the immediate cause of the explosion of nervous energy manifesting itself by the epileptic crisis, would afford, if proved, a probable explanation of the path of the ocular reflex through the intimate connection of the cerebral and sympathetic nerves which exists in and around the eye, and their mutual physiological and pathological relations.

Much time and space might be occupied in quoting from various authors reports of cases of epilepsy dependent upon eye-strain and cured by removal of the same. D'Abundo claims to have cured it by correction of astigmatism. Elliot, Colburn and Frothingham by convex glasses, and Stevens by operative measures in

heterophoria. Wood says in a discussion of the "Treatment of Epilepsy by Tenotomy of the Ocular Muscles," in the "New York Med. Journal" of July 7 and 14, 1894: "Anomalies of the muscles alone may produce epilepsy by acting as a reflex irritant. It is true that some cases of epilepsy are cured by muscular treatment when there is true heterophoria. In cases in which epilepsy is to be attributed to loss of ocular equilibrium, other symptoms of the latter are not wanting". Pechdo, Fumagalli and Galezowski report cures by enucleation. It is proper to remark in this connection that cases of epilepsy have been cured by a variety of trivial operative measures where the results must be attributed to the influence of suggestion. After making due allowance for such influence and for too enthusiastic opinions, and for errors of judgment and of diagnosis, many cases remain too well authenticated to admit of doubt.

If the preceding arguments are worthy of consideration, a single case will carry as much weight as many in establishing the possibility of such a cause for epilepsy, and of emphasizing the importance of examining the eyes in all cases where the cause and the remedy are not apparent. If this most serious and frequently incurable disease can thus be relieved even occasionally we shall be guilty of criminal neglect if we omit such examination. Dr. F. Park Lewis, in the "Eye, Ear and Throat Journal" for January, 1895, reports the following case of epilepsy dependent upon

eye-strain: "A bright young man of eighteen or thereabouts, was sent about two years ago to a military school where he was successful in his examinations and greatly interested in his work. The amount of mental work required was rather unsual, however, and the discipline rigid. Without any warning, he was one day taken with a genuine epileptic seizure. This was shortly followed by another and again another, so that it was necessary for him to return home. He was placed under the care of a prominent neurologist and treated generously with bromides. In spite of care and treatment, the attacks decreased neither in frequency nor severity, until his parents, having heard of eye-strain as a possible cause of convulsions of this character, brought him to me that he might have a comprehensive ocular examination. Tests under atropine showed a small degree of hypermetropia with a quarter of a dioptre of vertical astigmatism. He had, however, marked esophoria with weakness of the external recti. He was given appropriate prisms, operative measures being held in reserve. Surgical interference has never been resorted to, and from the time of wearing glasses until now—about a year ago—he has never had an attack."

Dr. Lewis remarks: "Other cases of like character might be given in which high degrees of exophoria and also astigmatism have been the exciting causes of epilepsy. In one instance, whenever the accommodation was relaxed with atropine, all convulsive tendency im-

mediately disappeared and did not return while the mydriasis was maintained. . . . . To fail to recognize the reflex nature of these cases is to shut our eyes in the only direction from which we may confidently look for relief. The fact that all epilepsies have not a reflex origin, or the equally palpable truth that muscular insufficiencies and other nerve strains exist without causing disturbances, either epileptic or otherwise, is no argument against the conclusion that I have drawn, and only leads to a plea for a more careful differentiation of symptoms and their probable cause in diagnosis, and a greater hesitancy in resorting to bromides or any palliative measures which may be of doubtful efficacy in the one set of cases, and must be of positive injury in another more complicated, and less easy to diagnose epileptic group."

Various eye symptoms have been noticed during an epileptic attack, such as spasm of the orbicularis and deviation of both eyes toward one side, and rolling movements. The lids may be open or closed, and the conjunctiva is insensitive. The pupils may be contracted at the beginning of the attack, but this is not invariable. Dilatation with inaction to light comes on simultaneously with the cyanosis, and continues until signs of consciousness begin to be manifested, when the mydriasis ceases. Afterwards there is alternate contraction and dilatation, changing every few seconds during an interval of several minutes. Frequently in the in-

terim between attacks there occur variations in the size of the pupils without apparent cause.

CHOREA.

Although pathological changes in the brain and spinal cord have been found in many cases of chorea, they vary so much in situation and character and are so frequently absent, that no definite conclusion can be drawn from them as to the cause of the affection. Hence it is classed among the neuroses. An hereditary neuropathic temperament usually underlies the disease, other members of the family frequently suffering from insanity, epilepsy or hysteria. It is much more frequent in the female sex, and during the impressionable age of childhood. Fright is the most frequent exciting cause where the predisposition exists. Cases have been attributed to intestinal worms, and to irritation of a peripheral nerve.

Eye-strain should be remembered as a possible factor, although the evidence of such an influence rests upon a much more slender basis than with epilepsy. Ranney, of New York, reports in the "Medical Record" for May 12, 1894, a series of cases of chorea all of which were cured or much benefitted by graduated tenotomies. Stevens attaches much importance to errors of refraction as a cause of chorea, and Gould claims to have cured one case by the use of convex glasses. This view is strongly opposed by other oculists of eminence.

The committee of the N. Y. Neurological Society, who were appointed some years ago to investigate Dr. Stevens' claims, came to the conclusion that they were not supported by facts.

De Schweinitz, of Philadelphia, analyzed 227 cases of chorea, in which he found either hypermetropia or hypermetropic astigmatism to exist in 76 per cent., and myopia and mixed astigmatism in a few of the remainder. At first sight this would seem to afford strong corroborative testimony in favor of the relation of cause and effect between chorea and refractive errors. When, however, we recall the fact that exactly the same, viz., 76 per cent. of all children in the elementary schools have hypermetropic refraction, the evidence seems as strong that chorea causes hypermetropia as the reverse.

Although the facial muscles are affected in nearly all cases, the ocular muscles are rarely involved.

Lifting of the eyebrows, closure of the lids and rolling of the eyes increasing on excitement and ceasing during sleep have been observed in this disease. Dilatation and inequality of the pupils may be present. Nystagmus occurs rarely, and points to a cerebral lesion as the cause both of the nystagmus and of the chorea. Thus its presence would be of diagnostic importance as indicating that the latter was not a simple neurosis.

NEURASTHENIA AND HYSTERIA.

Eye-strain is a direct cause of neurasthenia and an indirect cause of hysteria. The former is not a dis-

tinct disease, but a condition of nervous exhaustion which may be due to a variety of conditions both mental and physical, or it may be a direct sequence of definite diseases both functional and structural. It is not necessary to repeat in this connection what has been said in reference to the large expenditure of nervous energy required in *physiological* vision, and it needs no additional argument to emphasize the depressing influence of conditions demanding unusual nervous effort such as necessarily attends the visual act when errors of refraction or weakness of the external or internal eye-muscles exist. Here again we recognize the "vicious circle" of cause and effect, and will expect to find, as we do, all forms of asthenopia developed and intensified in neurasthenia. There is easy fatigue of the eyes and of the visual centres and anaesthesia of the retina; accommodation and convergence are also weak. These conditions explain the indistinctness of near vision and the pain attending it, and the necessity for convex glasses in those who have not previously required them, as well as the frequent change of glasses which those already dependent upon them require. The retinal and central exhaustion produces impairment of distant vision, frequent alterations of the visual fields and dilated and changeable pupils. Romberg called attention to an unusual symptom which he considered peculiar to neurasthenic individuals, viz., an inability to close the eyes completely when standing with the

feet close together, because of weakness of the accommodation and spasm of the sympathetic. Loenwerg states that this symptom occurs on standing, without regard to the position of the feet.

Certain peculiar visual disorders occur in neurasthenia, but are more frequent in hysteria, and, therefore, they will not be separately discussed here. There is an intimate relation between neurasthenia and hysteria, for the former underlies a large proportion of functional nervous diseases, including hysteria, and is often the direct cause of them.

HYSTERIA.

As with epilepsy and chorea, we recognize in hysteria also an hereditary neurotic tendency which is intensified by poor health, anaemia and any depressing influence such as eye-strain, which thus may be enumerated among the causes of hysteria. Therefore the correction of muscular and refractive errors is an important factor in the treatment of hysterical affections.

To reverse the subject, we recognize many functional ocular affections manifested in the domain of both the cerebral and the sympathetic nerves as a result of hysteria. A general characteristic of such disorders is suddenness of onset, erratic course, often a sudden disappearance, and the absence of apparent adequate cause of ophthalmoscopic changes or other evidence

of pathological basis for the symptoms exhibited. Such affections may, for convenience, be classified as:

a. Muscular disorders.
b. Disorders of sensation.
c. Disorders of secretion.
d. Visual disorders.

a. MUSCULAR DISORDERS.—Under this head may be mentioned twitching and spasmodic closure of the lids and spasm of accommodation. The latter produces apparent near-sightedness, indicated by more or less indistinctness of distant vision and an inclination to hold objects nearer the eye in reading, writing, etc. Spasm of accommodation is not at all characteristic of functional nervous troubles, for it occurs frequently in connection with errors of refraction, but hysteria and neurasthenia increase the liability to it when refractive errors exist.

Paralytic affections occasionally occur, among which hysterical ptosis is the most common form. The expression "hysterical ptosis" is rather misleading, however, for most cases are due rather to a slight spasm of the orbicularis than to a real weakness of the levator palpebrae. This is demonstrated by the circumstance that when the patient is told to look up she bends her head backward, and if the head is held, the spasmodic contraction of the lids is manifestly increased. In other cases paralysis of the sympathetic supplying the un-

striped muscular fibres of Mueller may explain the slight drooping of the lids.

It was positively asserted by Charcot in 1892 that "nystagmus is never found in hysteria," and Gowers, in the latest edition of his treatise on nervous diseases, lays special stress on the importance of nystagmus as a diagnostic sign, indicating the existence of "more than a functional disturbance of the brain or cord."

A case reported by Dr. Sabrazes in the "*Semaine Medic.*," September 26, 1894, seems to prove that these statements of Charcot and Gowers are not to be accepted without reservation. The patient had nystagmus without indication of any lesion of the visual apparatus, associated with manifest indications of hysteria. Hypnotic suggestion caused the immediate disappearance of all symptoms of disease.

Frequent variations in the size of the pupil without apparent cause was mentioned as a manifestation of neurasthenia, and it is also one of the recorded symptoms of hysteria.

b. DISORDERS OF SENSATION in and around the eyes as a manifestation of hysteria are frequent and varied. Pain occurs either spontaneously or with efforts of accommodation. Tenderness in the ciliary region and anaesthesia of the skin of the lids and of the conjunctiva and cornea are common.

c. VASO-MOTOR and other SYMPATHETIC DISTURBANCES in the eye are evidenced by disorders of secretion such as epiphora without irritation or emotion, and chromidrosis, or a coloration of the skin of the lids in consequence of an abnormal formation of pigment by the sweat glands.

d. VISUAL DISORDERS have been frequently noticed in hysteria. There may be slight impairment of vision or total blindness. Hysterical amblyopia, as it is called, is of sudden onset and unaccompanied with ophthalmoscopic changes or evidences of cerebral lesions. It may affect both eyes, but more frequently it is monocular. The reaction of the pupil to light may be retained or lost. If retained it indicates a cortical affection, probably a vaso-motor disturbance of the circulation. Hysterical amblyopia is usually of transient duration and sudden in its disappearance. It is often surprisingly improved by lenses of various sorts, convex, concave or prismatic. The explanation of such improvement must be an increased innervation caused by the use of the lenses.

Monocular amblyopia with retained pupillary reflex is strongly suggestive of simulation, and the differential diagnosis between simulation and hysteria is difficult. Apart from other hysterical manifestations, the test for diplopia by means of the stereoscope and prisms will afford valuable aid if carefully conducted. Binocular vision is essential for uniting the two pictures of

the stereoscope into a harmonious single image. Hence if the patient with alleged monocular amblyopia sees clearly with the stereoscope, the simulation of blindness of one eye is exposed. A prism of 6° or more held with its apex upward before one eye will produce a double image (one above the other) of a candle or gas-flame across the room *if both eyes are used in seeing.* The effect of prisms upon the visual lines can also be noted where it is not desirable to trust the statements of the patient. If a prism of 6° to 10° is placed before one eye with its base out, while the patient's gaze is directed towards an object a few feet distant, the eye behind the prism will make a movement inward (in the direction of the apex of the prism) to avoid the diplopia caused by the prism, unless that eye is blind or nearly so. The detection of the fraud is more difficult when the patient asserts that the sight of one eye is deficient, but not entirely lost. We must then in conducting the test endeavor to deceive him as to which eye is being tested. If he is able to read fine print with the amblyopic eye while he imagines he is using the sound eye, the simulation is apparent. Such a test may be conducted in the following manner:

By the aid of Snellen's or Jaeger's test-types we ascertain the smallest print he can read with the sound eye. Then, handing him a spectacle frame having a plain glass before the sound eye and a strong convex glass before the supposed amblyopic eye, we ask him to

read again. Later, under pretext of having neglected to note the size of the print previously read, or for other plausible reason, we repeat the test, this time reversing the lenses so as to bring the convex glass before the sound eye. If he now reads the same print at the former distance, the diagnosis is established.

Monocular diplopia is sometimes an hysterical manifestation and is usually caused by a spasm of accommodation, as is evidenced by the fact that the diplopia disappears under the use of atropin for the relaxation of accommodation. Dr. Lagrange reported to the "Medical and Surgical Society of Bordeaux," in November, 1894, a case of hysterical monocular diplopia which was unusual, in that it was evidently due to a cerebral condition, and in which the diplopia was cured by hypnotic suggestion. In this case the diplopia was uninfluenced by the use of atropin.

There is a peculiar form of the visual field which Charcot considers characteristic of hysteria, especially when it is unilateral. Excentric vision gives us the faculty of orientation, as it is called, which is of exceeding value. If, in walking, for instance, we were unconscious of all objects save those directly before us, we would not only lose much of the pleasure of seeing, but would be continually exposed to danger, for we would not notice the approach of objects on either side; neither could we appreciate the relative position and direction of objects. This is well illustrated in the case

of retinitis pigmentosa, where, although central vision in a limited area may still be preserved to such an extent that the patient is able to read fine print, yet be incapable of guiding himself alone.

Certain diseases produce characteristic alterations in the form and outline of the visual field. For instance, retro-bulbar neuritis causes a central scotoma, or a loss of vision characterized by a black spot in the centre of the field. In atrophy of the optic nerve there is a concentric narrowing advancing very irregularly from the periphery, often commencing from the temporal side. Sometimes the field is quite uniformly restricted on all sides, and, again, large gaps appear in one direction while the boundaries may be nearly normal in all others. Glaucoma produces a narrowing, usually manifested first on the nasal side. Hysteria sometimes produces a concentric contraction with a nearly uniform outline in all directions. This peculiar form of the visual field, existing without ophthalmoscopic findings, is very characteristic of hysteria, and hence is worthy of remembrance as affording a valuable diagnostic point in certain cases when a suspicion of cerebral disease exists. It may be associated with more or less impairment of central vision, and more or less loss of the color sense.

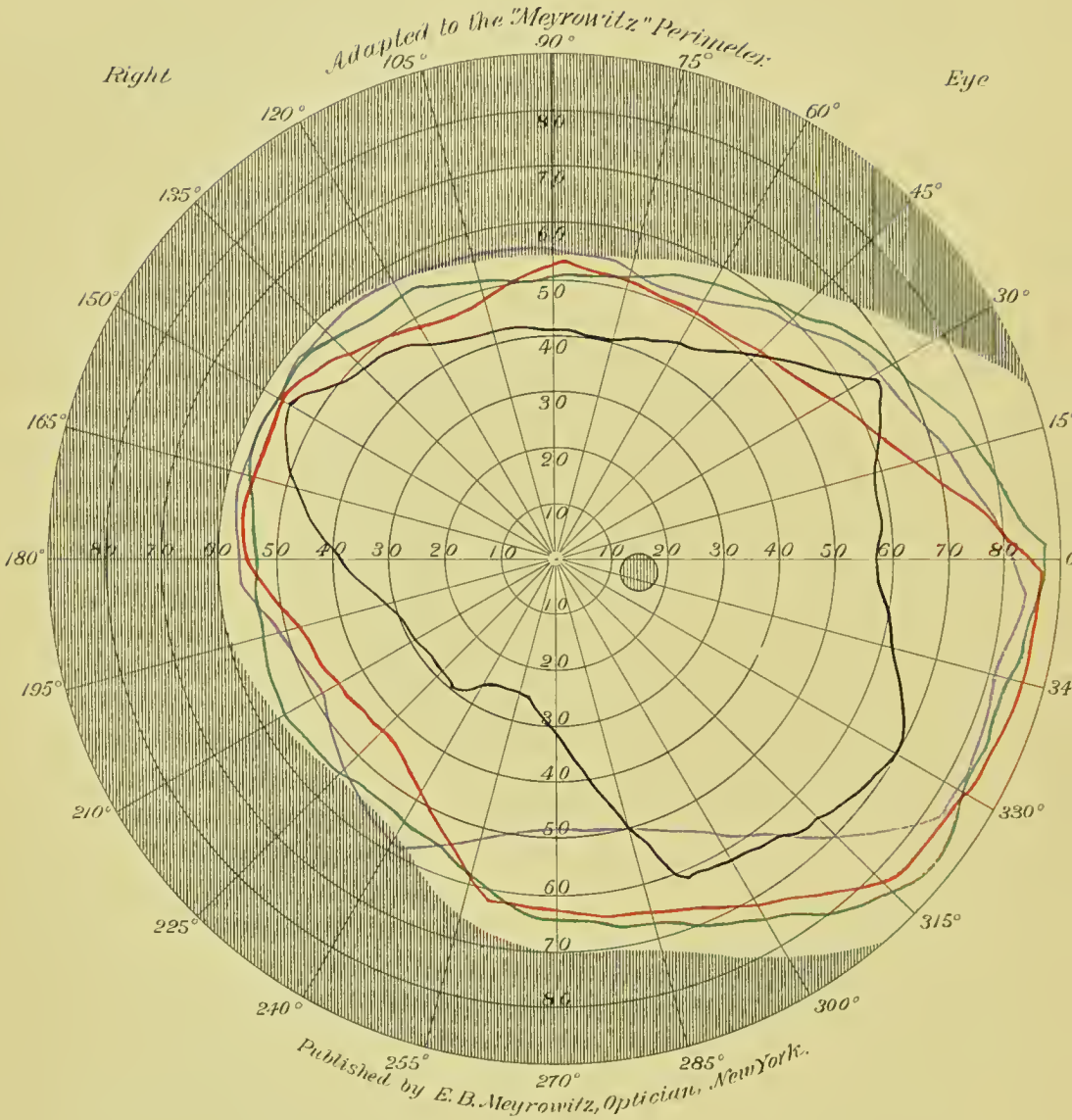
The following case is cited by way of illustration.

Addie C., a sensitive, nervous child, fourteen years old consulted me in February, 1896, complaining of headache, indistinctness of vision, restless dreamful sleep, and a variety of nervous symptoms which were apparently associated with the approach of puberty. She had never menstruated. Two years previously I had examined her and found slight hyperopic astigmatism in each eye with some amblyopia of the left. With glasses prescribed at that time the vision of the right eye was $\frac{15}{15}$, and that of the left $\frac{15}{30}$.

These glasses she had worn constantly and satisfactorily until shortly before consulting me in February, 1896. At that time v.o.d.$=\frac{15}{30}$ and v.o.s.$=\frac{15}{50}$, and could not be improved by glasses. She could not read print finer than Sn. No. 1. After paralyzing the accommodation with atropin distant vision was the same, and all lenses were again refused. Ophthalmoscopic examination showed nothing abnormal. The visual fields were concentrically contracted, with somewhat irregular outlines.

Under treatment for her general health, rest of the eyes and outdoor exercise, there was gradual improvement both in central vision and in the outline of the fields until August 5, when there was a slightly increased contraction of the latter. This was only temporary, however, and, on the second of September, she again accepted the lenses which corrected the slight astigmatism previously demonstrated, and vision of the

PLATE III



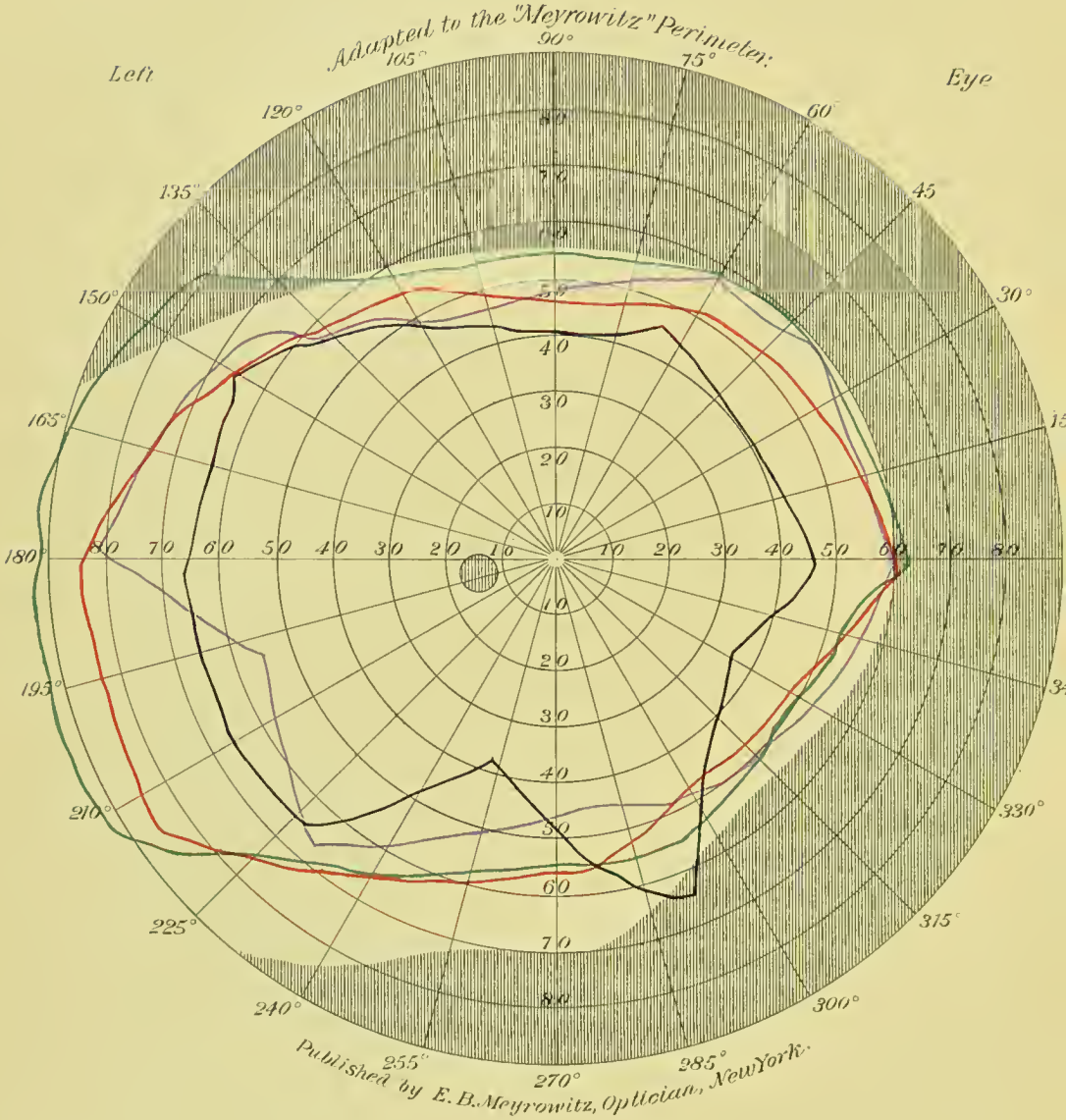
March 7th '96

June 5th '96

Aug. 5th '96

Sept. 2nd '96

PLATE IV



March 7th '96

June 5th '96

Aug. 5th '96

Sept. 2nd '96

right eye was again $\frac{15}{15}$ and that of the left $\frac{15}{30}$, the visual fields were practically normal as is shown by the appended charts (Plates III and IV), and she read .5 easily.

There are certain well-determined areas in which the various colors are perceived. The outlines of the color fields differ from those for form, and vary with the individual colors (see chart p. 115).

The same incongruity of manifestation which has been found in other hysterical affections is noticeable in the outlines of the various color fields. There may be complete loss of color perception, or the form of the fields may exhibit entire lack of uniformity as compared with the normal standard. With organic affections we notice irregular contractions of the field for form, and regular contractions of the color fields, while the reverse, viz.: uniform concentric narrowing of the field for form, and irregular, unsymmetrical contraction of the color fields occurs in hysterical affections.

Certain ocular symptoms have been recorded in connection with other functional nervous diseases, but, as they possess no diagnostic significance, they are not pertinent to the objects of this discussion.

LOCAL OCULAR REFLEX NEUROSES.

In the introduction to this chapter functional nervous diseases were divided into general or systemic, and local. It remains to consider the relation of ocular affections to local neuroses.

No argument is needed to convince the ophthalmologist, or the general practitioner, who is conversant with current medical literature, of the frequent dependence of headache of various sorts upon eye-strain. The cure of headache, often of long standing, by the correction of errors of refraction or of heterophoria is a matter of every day experience with the specialist. These two sources of eye-strain are so often associated that it is difficult to determine their relative importance, some writers attributing the greater influence to difficulty of accommodation and weakness of the extrinsic muscles, and others to refractive anomalies. To one or all of these conditions very many cases of headache are due. This fact should be borne in mind, and a thorough examination of the eyes should be made in all cases of frequent or continuous headache where the cause for the same is not evident, or where ordinary treatment is not satisfactory. It has been the experience of the writer that in these cases the patient frequently has not been conscious of any visual defect or asthenopic symptoms, yet examination has disclosed a muscular or refractive error, which has been demonstrated to be the cause of the headache, because correction of the former has been followed by disappearance of the latter. Such cases emphasize the importance of ocular investigation, even in the absence of eye symptoms.

Headaches from eye-strain present a variety of forms. Sub-occipital pain, suggestive of congestion of

the base of the brain, and symptoms of general cerebral hyperaemia are common; also dull pain over the eyes or behind them, or extending from the eyes to the vertex or occiput, and neuralgic pains in and about the eyes.

Hemicrania or migraine, popularly called "sick headache," frequently follows over-use of the eyes. It is probably an expression of general nervous exhaustion from over-exertion of weak organs, and the numerous visual phenomena, such as amblyopia, scotoma, hemianopic defects, phosphenes, *muscae volitantes*, etc., point to an irritation of the cortical visual centre. These visual symptoms are so pronounced in many cases as to give rise to the special classification of "ophthalmic migraine," or "scintillating scotoma." It is characterized by dazzling, luminous vibrations occurring in the right or left visual field of both eyes, with more or less complete homonymous hemianopia on the same side. The reactions of the pupil are preserved, affording another evidence of the cortical situation of the disturbance. The attack may or may not be accompanied with pain and nausea, but the absence of pain is the exception. It admits of but one explanation, viz.: a vaso-motor disturbance of the visual area of the cortex of the occipital lobe on the side opposite to the affected visual field. It may be a reflex ocular neurosis, but it frequently results from other causes, and is sometimes a manifestation of hysteria. Pure scintilating scotoma,

without fully-developed migraine, is comparatively of frequent occurrence in brain workers, and is not of serious import.

Visual symptoms occur in fully one-half of the cases of migraine, and their existence is of importance in making a differential diagnosis between the latter affection and supra or infra orbital neuralgia. Photophobia, lacrymation and conjunctivitis occur with both disorders. We may have either contracted or moderately dilated pupils with migraine, showing either spasm or paralysis of the sympathetic. These phenomena are absant in pure neuralgia, and in the latter, pressure upon the nerve is painful.

A severe case of migraine cured by correction of compound myopic astigmatism was reported by Noyes.

Dr. O. Landman in a recent journal "attempts to determine the character and behavior of headaches dependent upon an uncorrected error of refraction. According to him, migraine is only rarely caused by error of refraction, even when it is accompanied by this. It is especially uninterrupted headache which is caused by some ocular defect. It is more frequent in women than in men, and may generally be localized. Uninterrupted diffuse headache is usually caused by a general disturbance. If the patient complains of headache in the eyes, or of pain extending from the eye-balls to the head, or surrounding the eyes after work, or situated behind the eye-balls, or of exasperated attacks in the

evening in general, of painful symptoms yielding to repose, it is probable that the cause resides in the eyes. The author thinks that it is refraction and not a lack of equilibrium of the motor muscles of the eye which produces headache. Two hundred cases of ocular headache present the following subdivisions of localization: eye-brows, 41 per cent.; top of head, 20 per cent.; occipital, 12 per cent.; occipital frontal, 8 per cent.; temporal, 8 per cent. In a single case, the headache was general. The forehead and the vertex, and the occiput and the vertex were each thirteen times the seat of pain; occipital headache was frequently accompanied with stiffness of the nape of the neck."

Ocular vertigo may be mentioned in this connection, although we are discussing especially neuroses resulting from functional ocular diseases. It is not of frequent occurrence, but sometimes it is met with as a reflex nervous manifestation dependent upon paralysis of one or or more of the ocular muscles. When the effort is made to move the eye in the direction of the paralyzed muscle, diplopia and erroneous projection of the visual field results, the proper relation of objects is lost, and vertigo occurs. The trouble is usually of transient duration, for the sensorium soon learns to correct the false impressions received, but its recognition is important to prevent errors in prognosis, and useless and perhaps injurious treatment.

Insomnia and even mental aberration have been ascribed to eye-strain. Baker, of San Diego, Cal., reports in "Southern Cal. Prac." for January, 1893, a case of mental derangement in a woman 46 years of age which was promptly relieved by correction of mixed astigmatism. Baker, of Utica, reports in "American Journal of Insanity," April, 1893, several cases of "psychalgia" relieved by correcting errors of refraction.

Herren, of Jackson, in "Ophthalmic Record," January, 1893, reports recurring attacks of sneezing cured by correcting ametropia. Miles reports in the "Weekly Medical Review," 1884, 107 cases of functional nervous diseases in which there were frequent sneezing, nose-bleeding, catarrhal symptoms and itching and tickling sensations, which were either much relieved or entirely cured by the wearing of suitable glasses.

Nausea is not infrequently caused by using the eyes where there exists an uncorrected ametropia or muscular weakness. Neuschueler reports a cure of toothache by prisms. (Knies). Pains in the teeth frequently accompany the ciliary pains of keratitis, iritis and cyclitis. Cases of ocular reflexes to distant parts might be multiplied, but sufficient evidence has been offered to emphasize the fact, the remembrance of which may be of service occasionally to the intelligent and progressive physician.

Organic affections of the eye are not without influence as sources of reflex phenomena, but such influence usually is readily recognized and does not need special discussion. It is, for the most part, explained by the depression, mental and physical, which is entailed by pain and loss of sight.

One practical deduction from the preceding discussion is that many functional nervous affections are undoubtedly dependent to a greater or less degree upon eye-strain, and are relieved or radically cured by correction of errors of refraction and of heterophoria. Errors of not more than .25 D. frequently cause annoyance in persons of a sensitive nervous temperament, and their correction is followed by a disappearance of the symptoms. Apparently stupid and backward children often appear so because of defective vision.

On one occasion the writer witnessed relief following the prescription of as weak a glass as .12 D. The fact that such small errors are frequently endured without detriment, is no reason for always ignoring them, nor does it invalidate the teaching of clinical experience in other cases. The same conclusions have been reached in regard to sources of nerve strain in other organs, considerable departure from a normal standard being often endured without noticeable effect, and, on the other hand, apparently insignificant disorders being attended with serious reflex manifestations.

What has been demonstrated in regard to errors of refraction is also true of muscular defects. "Latent exophoria or esophoria of 2° to 4° is often physiological, and requires no treatment." (Maddox on Ophthalmological use of Prisms). Higher degrees frequently diminish after wearing corrective lenses. When they occasion annoyance they demand treatment either by exercise, tenotomy, or by the wearing of prisms or the decentration of lenses. Slight vertical deviations more frequently cause neurotic symptoms than do lateral ones of the same degree. I have repeatedly had gratifying results from the use of a vertical prism of one-half a degree.

If it could be positively determined in a given case that nervous affections were caused or aggravated by such ocular disorders as have been mentioned, it would be of inestimable value. Such data for an absolute diagnosis do not always exist, when pronounced subjective symptoms connected with the use of the eyes are not manifested. In doubtful cases rest of the eyes, with or without temporary paralysis of accommodation or temporary use of prisms, will solve the problem either by affording relief to the symptoms, or by demonstrating the existence of such pronounced errors as unhesitatingly demand correction, or that even slight errors are not well borne. Relief may be immediate or gradual following treatment of ocular disorders, and much judgment and experience is required to decide

the question of the influence of eye-strain in many obscure cases. This factor in the aetiology and perpetuation of functional nervous affections should ever be borne in mind by the ophthalmologist, the neurologist and the general practitioner.

Another deduction from this discussion is that examination of the eyes is of great value in the differential diagnosis between functional and organic nervous diseases.

CHAPTER VIII.

THE RELATION OF AFFECTIONS OF REMOTE ORGANS TO OCULAR NEUROSES.

Functional ocular disorders due to distant and circumscribed foci of irritation, either functional or organic, will now be considered. An inquiry into the mechanism and paths of reflex action, both physiological and pathological, will enable us to understand the subject more clearly and to appreciate its discussion.

For the production of reflex action three factors are necessary: first, an afferent impulse starting from the periphery, second, a reflex centre, and third, an efferent impulse starting from this centre and extending to the part where the reflex action is manifested. Two varieties of physiological reflex action are familiar, under the designation of the skin reflex and the tendon reflex. Irritation of the skin of the foot, for instance, produces flexion, and a sharp blow on the patella tendon extension of the leg. In these cases the path is through the cerebro-spinal nervous system to and from the reflex centre of the cord.

An afferent impulse may travel upward to the brain producing a sensation which again is reflected to a distant part or organ. In the feeling of faintness or

nausea from an unpleasant sight, for instance the efferent influence is transmitted through the pneumo-gastric, and in blushing, a vaso-motor influence is directed to the capillaries of the face. A sensation of pain may be felt in a remote locality by reflex influence emanating from a diseased part, or by simple suggestion, as by concentrating the attention upon a given portion of the body.

The functions of organic life are maintained through the agency of the unstriped muscular fibres in the viscera and blood-vessels under the control of the sympathetic system. Respiration, circulation, secretion, etc., are reflex acts which, as well as the general nutrition of the body, are regulated and dominated by the reflex centres in the medulla and the higher cerebral centres. While the sympathetic nerves contain motor fibres in large proportion, they also transmit afferent sensory impressions from the viscera, and the intimate connection between the cerebro-spinal and the sympathetic or, as Foster calls it, the splanchnic systems of nerves, permits ready transmission of impressions from one system to the other. The vaso-motor nerves, by which the calibre of the vessels is controlled, and the blood supply regulated according to the physiological requirements of nutrition, secretion and general functional activity, are controlled by the vaso-motor centres in the cord, and by the higher centre in the medulla.

Thus we see that reflex action may take place through the medium of either the cerebro-spinal or the sympathetic systems, singly or jointly, and may be manifested as psychic, sensory, motor, vaso-motor and trophic phenomena. The effect depends upon the strength of the initial impression and the condition of excitability of the centre. There are well-defined channels for the conduction of the ordinary physiological reflexes, but the structure of the central nervous system permits of the transmission of the efferent impulse along various paths, and we find pathological reflex action to be either a diminution or increase of physiological action, or a perversion of the same. There are reflex centres situated all along the spinal cord which receive and transmit sensory and motor influences throughout the areas supplied by the corresponding spinal nerves. Similarly, there are sympathetic ganglia on either side of the spine, along its whole extent, anastomosing freely with all the spinal nerves. The spinal reflex centres communicate freely by anastomosing fibres, and, as has been mentioned, are connected with the higher reflex centre of the medulla, and controlled by it. The latter probably is influenced by still higher cerebral centres, the exact location of which has not been definitely determined. This is rendered probable by the reflex disturbances following psychical impressions of fear, surprise, disgust, pain, etc. The spinal sympathetic ganglia are continued upwards into the

cranial cavity, and through the cervical, carotid, Gasserian, ophthalmic, otic, and spheno-palatine ganglia, are in communication with the cranial nerves.

Thus we find an unbroken nervous chain extending all over the body to its remotest parts, with free anastomoses between the different systems of nerves. Any sensitive surface, under pathological conditions, may give rise to reflex action of the most diverse kinds, and the location and nature of such disturbances is determined by the strength of the initial impression and by the condition of the reflex centres, the direction of the efferent impulse being along the "lines of least resistance." Suppose, for instance, an impression is received from a diseased point at a given spinal centre. The latter has numerous communicating lines in the direction of the sympathetic and also in the direction of other spinal centres. If the first centre is in normal condition, no efferent influence is excited to be transmitted along the nerves with which it is in special relation. The sensory influence is continued along one of the many anastomosing branches, and from centre to centre. Sooner or later, an unusually excitable centre is reached, or one having organs or tissues under its control which are in a condition of lowered vitality, or are morbidly impressionable. The deleterious influence is then manifested in one of the ways referred to, when under strictly physiological conditions it would still be inoperative. A lessened or exaggerated vaso-motor control

by the central ganglia may be the determining factor, and vaso-motor paralysis or spasm in the part finally reached will cause hyperaemia or anaemia, developing, if unchecked, into inflammation with exudation and perhaps a neoplastic growth, or causing exaggerated, diminished or perverted function. Reflex neuroses usually are vascular at first, and due to vaso-motor disturbances which induce, secondarily, functional and organic changes.

On the other hand, spasm or paralysis of voluntary muscular fibre may result through transmission of the impulse along a motor nerve of the cerebro-spinal system, or only a painful sensation may be experienced in consequence of a remote excitation.

Thus we understand how peripheral irritation from almost any point may reach the eye, and a knowledge of this fact may sometimes enable us to unravel a difficult case, and to find a remedy for a functional eye trouble through treatment intelligently directed to the original cause of the disorder. Here great care and discrimination is necessary to avoid confounding mere coincidence with cause and effect. The eye symptoms and the symptoms of the distant parts may both result directly from the same cause. Let us first examine some of the physiological ocular reflexes, and then inquire what are some of the functional disturbances of the eye which are recognized as due to reflex influences, and what pathological conditions have been demonstrated to occasion them.

PHYSIOLOGICAL OCULAR REFLEXES.

The contraction of the pupil upon the stimulus of the light and its dilatation following irritation of the skin are examples of physiological reflex action. In the former case the stimulation of the retina is conveyed to the habenular ganglion or the anterior tubercula quadrigemina, and thence an efferent motor influence is sent along the motor oculi. In the latter the peripheral sensory nerves convey the impression through the spinal cord to the corpora quadrigemina, and thence the motor influence travels down the upper cervical cord and along the cervical sympathetic to the carotid, the cavernous and the ophthalmic ganglia, and through the ciliary nerves to the iris. Winking and lachrymation from irritation of the conjunctiva or cornea are also reflex acts for the protection of the eye.

Pathological reflexes, or ocular neuroses, are very numerous and varied, and are of similar nature to those already enumerated in the discussion of general functional nervous diseases.

Numerous organic diseases are sometimes of reflex origin, but in the present discussion our attention will be confined to purely functional disturbances. It is not desirable to consider these various neuroses in detail, but a brief enumeration of the more frequent manifestations of reflex irritation may be helpful in preventing errors in diagnosis.

Where neurasthenia and hysteria could be excluded, the following conditions have been recorded by trustworthy observers, as caused directly by reflex influences emanating from distant foci of irritation, and cured by their removal: pain of various kinds in and about the eye, photophobia, lachrymation, nictitation, ptosis, lagophthalmus, temporary strabismus, myosis, mydriasis, anaemia and hyperaemia of the fundus from vaso-motor spasm or paresis, muscae volitantes, diplopia, amblyopia, scotomata and concentric contraction of the visual fields.

Nasal and dental reflexes are the most frequent, and when we consider the intimate nervous anastomoses between the nose and teeth and the eye, we are not surprised that such is the case. "The spheno-palatine ganglion sends branches to the nasal mucosa, and is in direct communication with the Gasserian ganglion at the sensory root of the trigeminus. The otic, ophthalmic, maxillary and superior and inferior dental ganglia are united in common through the sympathetic, giving a rich field of nervous network, in any part of which, transmission of any irritation sufficient to disturb its normal physiological functions could produce neurotic phenomena."

The intimate relation between the eye and the nose is shown by the physiological reflex actions of lachrymation following irritation of the nose, and of sneezing attending irritation of the retina by strong light.

Various nasal conditions have caused ocular neuroses by reflex action. A distinction must be made between such functional neuroses and affections due to extension of inflammation, or to the absorption of septic material by the blood vessels or lympathics. Certain inflammatory conditions of the structure of the eye are also at times of reflex origin from nasal diseases, but the definition of the term neuroses, as accepted in this treatise, precludes their discussion.

Various authors have reported ocular affections, such as those enumerated, associated with the following nasal disorders, viz.: polypi, hypertrophy of the turbinateds, abnormalities of the septum, and inflammation of the maxillary, sphenoidal and ethmoidal sinuses, which were cured by treatment of such conditions by galvanocautery and operations with the saw and snare.

The following cases reported by Dr. T. M. Stewart, of Cincinnati, in the "Eye, Ear and Throat Journal," are cited by way of illustration:

CASE FIRST.—"Mrs. B. applied for treatment, giving a history of constant dull aching in the left eye, gradually growing worse during the past three years. Glasses had been worn without relief. Aching in eye made worse during each attack of rhinitis, which attacks were frequent and caused by the least exposure. The left inferior meatus was quite patulous, but the middle turbinated body was swollen to the extent of touching the septum, and proved to be a genuine hy-

pertrophy. Reduction of the enlarged turbinated was followed by some relief, but not of sufficient amount to call the case cured. At a later visit upon deep exploration with a probe, we found an osseous spur on the septum pressing against the middle turbinated body. This growth was unsuspected and entirely hidden until after reduction of the enlarged middle turbinated. The growth was operated with the trephine, and no return of the headache and eyeache was noted for a period of three months. Re-examination showed a fibrous band of union between the cut surface on the septum and the middle turbinated. This was cut away and a dressing of borated gauze placed between the opposing surfaces. After it was removed and the parts healed, we again had the satisfaction of noting the disappearance of the head pains. Later the case presented itself at the clinic, with the report that for five months no return had been noted of the head pains. Previously to reporting the case here, inquiry was made by mail, and now sixteen months have passed with no return of the trouble."

CASE SECOND.—"Deep seated pain in the eyes on reading or sewing for even a few moments in a woman aged thirty-five years. Complains of pressure over the bridge of the nose and on top of the head. Catching cold would send streaks of pain from the bridge of the nose to the seat of the steady pressure on top of the head. Condition had existed for over two years.

"We found comparatively free breathing space, but both middle turbinateds were enlarged anteriorly, crowding the outer walls and the septum. Removal of the enlarged turbinateds by the forceps stopped the pressure on top of the head, but some pain on use of the eyes continued. Deep exploration revealed an osseous growth on the septum, and an adhesion to it of the posterior part of the middle turbinated. Removal of the growth and division of the adhesion cured the case, in that ten months have elapsed with no return of the symptoms."

Browne, in "Diseases of the Nose and Throat," reports a case of glaucoma cured by removal of a nasal polypus after iridectomy had failed to relieve. Goitre and Basedow's disease have been cured by the same means.

It is also worthy of note, both as confirming the causal relation of nasal and ocular affections, and as a warning to inexperienced operators, that similar ocular affections have followed operations on the nose.

A case in point occurred recently in my own practice. I removed an enchondroma from the right side of the septum. Vision in the eye on the same side declined from $\frac{15}{15}$ to $\frac{15}{30}$, without ophthalmoscopic changes in the interior of the eye, and with no other apparent cause. The impairment of vision lasted for two months and again became normal.

Musehold cured a case of exophthalmic goitre by removal of a hyperplastic growth of the inferior turbinated, and he reports five similar cases from the "*Deutsche Medicinische Wochenschrift*," Feb., 1892. He considers the disease a vaso-dilator neurosis. (Dr. White, of Richmond, in the discussion of nasal neuroses in Burnett's "Treatise on Diseases of Ear, Nose and Throat").

Trousseau claims to have cured two cases of blepharospasm, one of obstinate scotoma scintallans, one of mydriasis and three obstinate asthenopias by treatment of the nasal mucus membrane. Ziem reports a case of contraction of the visual field cured by restoring the outflow from the antrum of Highmore.

There seems to be some evidence that glaucoma is, at least in some instances, of reflex origin through the sympathetic nerves. Irritation of the sympathetic causes increased ocular tension, while its section causes diminished tension. The same results follow irritation or paralysis of the fifth nerve by communication with the sympathetic through the ciliary ganglion. On first thought one would expect to find minus tension following stimulation of the sympathetic, and the reverse when it is paralyzed. The explanation lies probably in a stimulation of the vaso-dilator fibres which attends reflex action. Noyes remarks that "neuralgia of the fifth nerve is potential, and nervous strain is a provocation of glaucoma." Ziem claims to have secured temporary

enlargement of the field in chronic glaucoma by galvano-cautery of the nasal mucous membrane, and Lennox Browne reports a case of glaucoma rapidly cured by removal of a nasal polypus.

The relation of dental affections, especially caries, to ocular neuroses is substantiated by the record of more numerous cures following treatment of the teeth than in the case of nasal reflexes. Amblyopia and amaurosis have been reported several times in such connection. Neuralgic toothache is sometimes a prodromal stage of glaucoma. Sous, in the "*Journal de Médicine de Bordeaux*," Nov. 20, 1893, reports the following case: "A young woman, twenty-one years of age, of a lymphatic constitution, but in perfect health, had vision reduced to $\frac{1}{10}$ and amplitude of accommodation much reduced. Both conditions returned to normal after a dental condition was cared for. He explained the case by reflex action of the superior maxillary division of the fifth nerve upon the ophthalmic branch of Willis."

Reflexes from the outer and middle ear to the eye are occasionally noted. Blepharospasm is the most frequent variety.

The influence of helminthiasis and sexual disorders in both males and females is frequent and needs no comment. Androgsky, of St. Petersburg, reports in Zehender's "*Klinische Monats blätter fur Augenheilkunde*," Stuttgart, Vol. 32, p. 263, 1894, two cases of chronic

spasm of the orbicularis muscle produced by the presence of tape-worm in the alimentary canal. Functional uterine affections are more influential in this direction than organic.

The foregoing discussion emphasizes the dependence of certain functional eye troubles upon reflex action emanating from distant sources of irritation. In the absence of functional or organic constitutional disease which would offer a probable explanation of such existing eye symptoms, and when no satisfactory cause for them is found in the eye or its appendages, and yet they do not yield to appropriate treatment, then a reflex origin is to be sought in the nose, the teeth, the ear, or in the digestive and sexual systems.

PART THIRD.

OCULAR AFFECTIONS OF TOXIC ORIGIN.

CHAPTER IX.

TOXIC AMBLYOPIA. CHRONIC RETRO-BULBAR NEURITIS.

Certain drugs, when administered internally, externally or hypodermically, are capable of causing ocular symptoms which may or may not be of serious import, but which it is important to recognize. They advise the observant physician that a remedy is not well borne, or that the patient possesses a peculiar susceptibility to it. A knowledge of these occasional occurrences is a check upon the injudicious and careless employment of such remedies, or when such drug effects are simply annoying and not dangerous, their recognition enables the physician to quiet unnecessary apprehension on the part of the patient or friends. Many poisons produce characteristic eye symptoms which are an important aid in the diagnosis of the substance which has been administered by accident or design, and thus may be not only a guide to treatment, but of prime importance in a medico-legal aspect. Again the existence of certain eye symptoms sometimes affords an explanation of obscure conditions in other parts of the body due to toxic influences hitherto unsuspected. Certain articles of food and drink, if improperly prepared, or when taken immoderately, or where a per-

sonal idiosyncracy exists, cause a condition of toxaemia which gives rise to ocular symptoms, the explanation of which is important, both as regards diagnosis and treatment. The same may be said of ptomaine poisoning. Certain avocations require the use of noxious substances which affect the eye or the vision, and their recognition is also important. It is not my purpose in the present discussion to enumerate every recorded toxic symptom that has been observed in the department of ophthalmology, but only those of diagnostic importance either as determining the cause and nature of the ocular manifestations, or as an aid in the diagnosis of constitutional disorders. Only those affections which are directly of toxic origin will be considered. Those due to uraemia or occurring in the course of diabetes, although in reality of a toxic nature, or those which are the indirect result of poisons which induce vascular changes, such as occur in chronic alcoholism or those consecutive to cerebral or spinal or cardiac lesions will not be discussed here. The reader is referred to other portions of this work for a consideration of such affections. The present topic may be classified as follows:

I. Toxic amblyopia.—By this term is ordinarily understood the disturbances of vision caused by the inordinate use of tobacco and alcohol. I shall also consider under this head similar disturbances of vision caused by various other substances.

II. Ocular Affections Following the Employment of Various Therapeutic Agents.

III. The Effects of Non-Medicinal Poisonous Substances and those Attending Certain Avocations.

IV. Ocular Affections due to Toxic Substances Contained in Certain Articles of Food and Drink: a. Fungus Poisoning; b. Ptomaine Poisoning.

V. Ocular Conditions During and Following Anaesthesia.

I. Toxic Amblyopia.

A. TOBACCO AND ALCOHOL AMBLYOPIA.

Each of these agents causes a disturbance of vision with nearly identical symptoms and they are, therefore, mentioned together. As will be indicated later there are slight differences in their manifestations, but the essential phenomena from which the diagnosis of the affection is made are the same. Usually both agents are factors in producing the disease, but either alone has occasioned it. Long continued and excessive indulgence in either alcohol or tobacco is necessary for its development. It seldom occurs in persons under thirty-five years of age, and it is usually older individuals with impaired nutrition who are thus affected. Much depends also upon the quality of the liquor and the tobacco used. There is more fusel oil in poor

whiskey, and more nicotine in the inferior and cheaper grades of tobacco, than in the more expensive varieties. Hence we find more cases of toxic amblyopia among the poorer classes.

Berry in "Trans. Oph. Soc. United Kingdom," Vol. VII, p. 91, estimates the amount of tobacco which causes amblyopia as from one ounce to half a pound or more weekly. The effect depends somewhat upon the manner of using tobacco. Smoking when the stomach is empty, and inhaling the smoke whereby it comes in contact with a large absorbing mucous surface, is especially injurious. Personal idiosyncrasy is also a very important factor influencing the development of toxic symptoms.

As most habitual drinkers also use tobacco it is difficult in most cases to determine the relative importance of each. In France the greater influence is assigned by many writers to alcohol, but most authorities consider tobacco as responsible for the larger number of cases of toxic amblyopia.

Among Orientals, where the use of the weed is almost universal, visual disturbance as a result is very infrequent. This immunity is ascribed to the mildness of the tobacco and to the way in which it is used. De Schweinitz says ("Toxic Amblyopias," 1896) "Turks while smoking cigarettes, are particular that no tobacco shall come in contact with the buccal mucous membrane, and, although they inhale the smoke vigorously,

they are not poisoned, because, according to Von Milligen, tobacco poisoning (and hence amblyopia) is possible only when nicotine in solution is brought in contact with the mucous membrane of the mouth and swallowed."

Smoking is more harmful than chewing, but Noyes, in his "Diseases of the Eye," p. 683, asserts that he has observed the disease as a result of chewing alone. Cases are also on record where it has been caused by the practice of "dipping" or rubbing snuff upon the gums.

In a tabulated report of 204 cases of retro-bulbar neuritis (the pathological condition which obtains in toxic amblyopia) he found 64 to be the result of alcohol, 23 the result of tobacco, and 45 were attributed to the combined influence of the two, so that more than one-half of the whole number resulted from one or both of these agents.

Uhthoff (see *Graefe's Archive*, Bd. XXXII, *et al.*) examined 1000 cases of alcoholic excess, in which he found 6 per cent. of amblyopia, and other 6.5 per cent. exhibiting the lesion of the optic nerve which is characteristic of toxic amblyopia, but in which the visual symptoms had not developed.

In the "Quarterly Journal of Inebriety," for Jan., 1893, Dowling, of Cincinnati, publishes a report of an examination of 150 employees in a tobacco factory, among whom 45 exhibited more or less evidence of

tobacco amblyopia. He thinks the inhalation of tobacco dust is capable of producing the disease.

It does not require expert skill or long experience to diagnose the affection. The ophthalmoscopic symptoms are absent or trivial until a late stage, hence the diagnosis is made from the subjective symptoms.

There is an acute, non-toxic form of retro-bulbar neuritis resulting from severe exertion, sudden chilling of the body, suppression of menstruation, rheumatism, syphilis or lead poisoning, or sometimes accompanying infectious diseases, especially measles, angina and influenza. In these cases it is evident that a condition of toxaemia underlies and causes the amblyopia, but not in the sense of the present discussion. These cases are sudden in development, and are accompanied with more or less pain in and around the eyes. The loss of vision may be complete, while this has very rarely been recorded in the case of tobacco or alcohol amblyopia. The acute form is more often unilateral, while the chronic toxic form is very rarely limited to one eye.

Dr. d'Oench, of New York, reported a case of toxic amblyopia due apparently to the abuse of alcohol and tobacco, in which within the short space of twelve hours, vision was reduced from the normal standard to the ability only to count fingers at two feet distance from the eye. (See "N. Y. Med. Record," July 14, 1894).

The patient usually exhibits a gradual failure of central vision constituting a central scotoma, a hiatus or black spot in the centre of the visual field, while the peripheral vision remains unimpaired. At first the vision for color fails, red and green are not as bright as formerly, and later are not recognized at all. Soon the perception of blue and yellow fails, and later, not even the form of objects is recognized, and a perfect blank in the centre of the visual field is left. The patient sees nothing directly in the line of vision, although objects at one side are seen with the usual distinctness. He, therefore, cannot see to read or write, or if the scotoma is not too large, the middle of a sentence or, perhaps, of a word is lost, while the two ends of the line are seen imperfectly. It is very rare for peripheral vision to be lost, so that complete blindness is not to be feared. Usually both eyes are equally affected. Patients often complain of an annoying sense of dazzling when in a bright light, and this may be the first symptom for which the individual consults the physician. Owing to this circumstance, the patient declares that he sees better in a dim light, and in some instances this is true to a limited extent, although, as a rule, the improvement is more imaginary than real.

PATHOLOGY.—The pathological basis of this condition is an atrophy of the bundle of fibres in the optic nerve which supplies the macula lutea and its immediate vicinity. This atrophy is the result of a slow and

slight interstitial neuritis, but the acute variety of retro-bulbar neuritis is designated as inflammatory, in distinction from the chronic toxic form in which there is no pain or other manifest signs of inflammation.

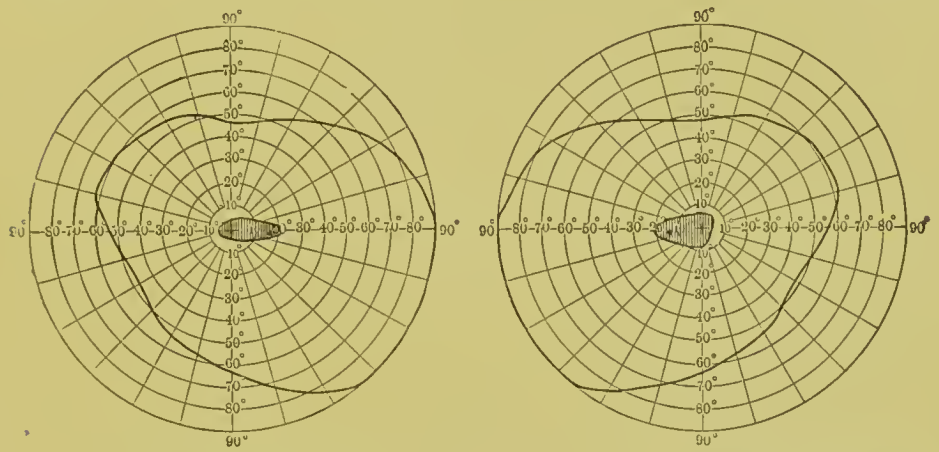
The situation of this atrophy is at first in the portion of the nerve lying in the optic canal, that is, behind the eyeball and in front of the chiasm, as its name implies. As the disease progresses, the atrophic process extends downward until it becomes evident at the optic disc, by ophthalmoscopic examination, at its lower temporal side, for it is at this point that the bundle of fibres designated enters the eyeball. The selection of this special bundle of fibres by these poisonous agents is an interesting and hitherto unexplained fact. It is not invariable, for cases have been reported where the peripheral fibres were affected, leaving the macular bundles intact, in which case, of course, the peripheral vision would be lost and central vision be retained. This, however, is of very exceptional occurrence, and is not pathognomonic of tobacco or alcohol amblyopia. There is no noticeable change in the appearance of the vessels of the disc or retina.

DIFFERENTIAL DIAGNOSIS BETWEEN TOBACCO AND ALCOHOL AMBLYOPIA.

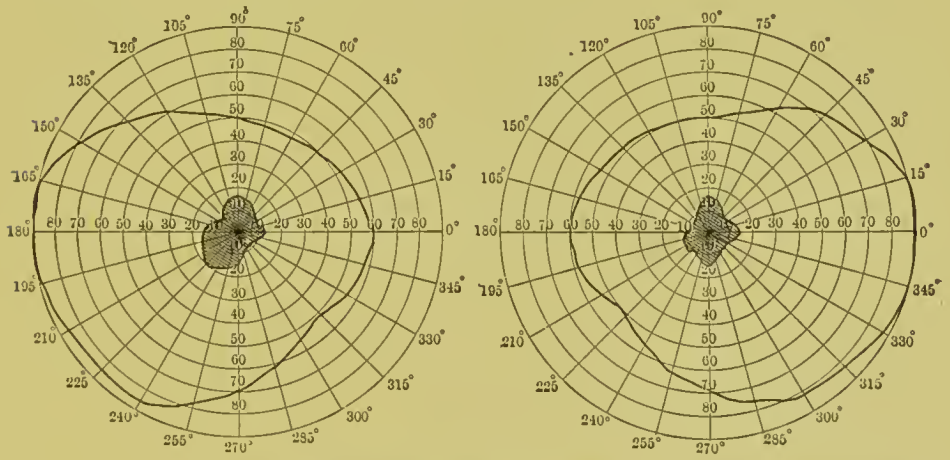
In alcohol poisoning the scotoma is central and always includes the point of fixation, but with tobacco the scotoma is very near but not at the point of fixation. This is probably not invariably the case. In

tobacco amblyopia the scotoma is usually of an oval shape, with the long horizontal diameter extending between the point of fixation and the blind spot.

The following charts of tobacco and alcohol scotomas are taken from de Schweinitz's "Toxic Amblyopia."



Tobacco Amblyopia; absolute central scotomas; peripheral boundaries of field normal in extent.



Typical oval scotomas from a case of tobacco amblyopia. The patient, aged sixty, had smoked five pipes of tobacco daily and an occasional cigar since he was nineteen; a moderate beer drinker.

With tobacco the amblyopia is oftener unilateral or differing in degree in the two eyes. With alcohol the pupil is dilated and the accommodation weak, while with tobacco we find a contracted pupil and spasm of accommodation. Paresis of accommodation may be one of the first disorders of vision of chronic inebriety, and should serve as a warning not to be disregarded.

The alcoholic form of the disease develops rather more rapidly than that due to tobacco, and the latter is less amenable to treatment.

In this connection certain other ocular phenomena accompanying intoxication with alcohol are worthy of mention, viz.:

Marked concentric narrowing of the visual field and diplopia.

Sudden blindness with merely quantitative perception of light, associated with dilated and irresponsive pupils, and with absence of any abnormal conditions of the fundus as seen with the ophthalmoscope, has been observed in acute alcoholism.

The differential diagnosis between true tabes and the simulation of that disease resulting from chronic alcoholism is aided by remembering that myosis is absent in the latter condition. Other symptoms of the real affection are also absent, but it is not the province of this treatise to discuss them.

B. RETRO-BULBAR NEURITIS DUE TO OTHER POISONS.

Toxic amblyopia with central scotoma has also been caused by the following substances, viz.:

STRAMONIUM.—Fuchs reports a case caused by smoking stramonium leaves for the relief of asthma. See Fuchs' "Text-book of Ophthalmology," p. 441.

BISULPHIDE OF CARBON, SULPHUR CHLORIDE, LEAD, AND PERHAPS CHLORAL.—Juler asserts that excessive TEA drinkers are predisposed to this disease and OPIUM, it is alleged, has also caused it.

ARSENIC has produced a form of retro-bulbar neuritis, and so has CANNABIS INDICA.

Hirschberg reported a typical case of toxic amblyopia with central scotoma and without ophthalmoscopic changes occurring in a girl of sixteen, upon whom IODOFORM was being used locally after a resection of the hip. A similar result was observed by Hutchinson during the internal administration of the drug in combination with CREOSOTE. This fact should be borne in mind, that the first indication of such poisonous influence may be recognized, in view of the extensive use of this drug as a topical application.

CHAPTER X.

OCULAR AFFECTIONS CAUSED BY VARIOUS THERAPEUTIC AGENTS.

Various drugs in addition to those already mentioned sometimes produce toxic symptoms, such as impaired vision, pupillary phenomena, disturbances of accommodation and of motility, and even organic lesions of various structures of the eye which it is desirable to consider.

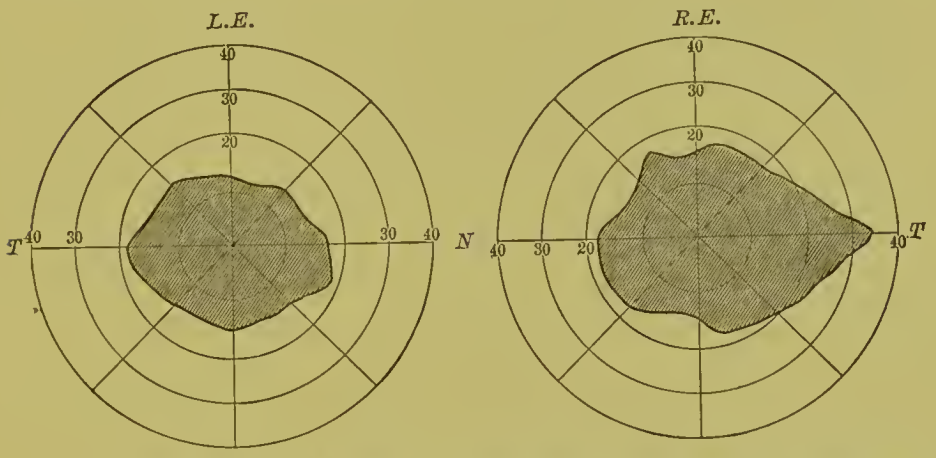
A. DISORDERS OF VISION.

Quinine Amaurosis.—Quinine produces a disturbance of vision exactly the reverse of that which has been described as due to the influence of tobacco and alcohol, viz.: a condition sometimes designated as telescopic vision. The periphery of the visual field is lost, and vision is restricted to a very narrow area in the centre, frequently of an elliptical shape with the long axis horizontal. The following charts illustrate the condition. They are taken from de Schweinitz. The shaded areas represent the limits of the field.

Usually there is, at first, more or less loss of central vision which sometimes amounts to complete blindness. Therefore we speak of quinine amaurosis in-

stead of amblyopia, by which latter expression we designate the slighter impairment of sight which results from the influence of tobacco and alcohol.

The amaurosis lasts from a few hours to several weeks. Many cases completely recover. In many others the peripheral vision remains permanently impaired, and in a few there is but slight improvement of central vision. This was true in a case that came under my



Visual fields in a case of quinine amaurosis three months after recovery from complete blindness.

own observation. In addition to the visual symptoms mentioned there may be hallucinations of sight, mydriasis, impaired accommodation, anaesthesia of the cornea, nystagmus, divergent strabismus and slight exophthalmus.

The amblyopia develops suddenly. Noyes says that it is always the result of large doses, and usually of repeated large doses, and that no cases have been

recorded where it resulted from small or even moderate doses. He does not define what he means by small or moderate but mentions one case that developed after taking four hundred and eighty grains in the course of twenty-four hours.

The inaccuracy of his assertion is demonstrated by a case reported by Pischl, of San Francisco, in the "Medical News" for July 20, 1893. Only thirty grains were taken during twenty-four hours. While central vision was but slightly affected, the fields were contracted to 5°. It will be remembered that the natural limits are from 45°to 90°, varying in different directions, being limited upward and toward the median side by the projection of the brows and the bridge of the nose (see chart of normal field, p. 115). After five weeks' treatment there was only partial recovery.

Cases of amblyopia have also been reported following the exhibition of fifteen, twelve, and even a fewer number of grains in twenty-four hours. The effect depends largely upon individual idiosyncrasy, and persons of a neurotic temperament are more susceptible to its toxic influence. Such cases as those to which allusion has been made ought certainly to teach caution in the use of the remedy, and it is important to remember that a person who has once suffered from quinine amaurosis is afterward much more susceptible to the toxic influence of the drug. Other alkaloids of cinchona, especially cinchonine and cinchonidine, occasion-

ally have the same effect upon vision, and it has followed the use of the tincture.

Claiborne, of New York, in the "Medical Record" for August 14, 1894, formulates his conclusions in regard to quinine amaurosis as follows:

"1. Quinine in toxic doses may produce blindness.

2. The duration of the amaurosis varies greatly.

3. The toxic dose is distinctly indeterminate.

4. The field of vision remains contracted.

5. Central vision usually returns to normal.

6. There is color blindness at first, color perception being ultimately restored in the central field.

7. The ophthalmological picture is that of white atrophy.

8. Experiments upon dogs show that there is atrophy of the entire optic tract.

9. The same experiments show that the cells of the cuneus are probably not affected.

10. Treatment is of no avail."

PATHOLOGY.—The ophthalmoscope shows a condition of ischaemia of the retina, which affords an explanation of the visual symptoms by the occurrence of sudden arterial spasm. Both veins and arteries are very much contracted. There are, as a rule, no ophthalmoscopic evidences of inflammation or exudation. Knies says that a slight exudation may occur in the macula lutea producing a cherry-red spot, very similar to the

appearance found in embolism of the central retinal artery. The optic discs appear paler, but their outlines are sharply defined.

De Schweinitz in the "Transactions of the American Ophthalmological Society," for 1891, p. 23, says: "The original effect is upon the vaso-motor centres, producing constriction of the vessels; finally changes in the vessels themselves are set up, owing perhaps to endo-vasculitis. Thrombosis may occur, and the result of all these is an extensive atrophy of the visual tract."

The amblyopia from quinine is usually associated with sudden deafness, but curiously enough, the latter effect has usually been attributed to hyperaemia instead of anaemia. In this connection the remarks of the editor of the translation of Gruber's work on diseases of the ear in the second American edition are worthy of mention. He says:

"Brunner calls attention to the contradiction existing between the statements of aural and ophthalmic surgeons concerning the effects of quinine. According to Kirchner and Roosa, large doses produce hyperaemia and extravasation of blood; while ophthalmologists state that it produces marked anaemia of all the retinal vessels. It must, however, be mentioned that in the experiments made by Gruber under Weber Liel's supervision, the temperature in the external auditory canal diminished after its employment, and hyperaemia of the canal or of the vessels of the malleus was never observed.

Similar results were obtained with salicylate of soda. The mitigation of auditory vertigo following its administration would in this way be explained by ischaemia of the labyrinthine vessels."

SALICYLATE OF SODA is said to produce visual symptoms similar to those which have been mentioned as a result of quinine. Complete blindness has been observed after taking eight grammes within a space of ten hours. It was associated with marked mydriasis and with normal ophthalmoscopic appearances. The retinal veins were well filled. After ten hours the patient could count fingers, and vision was completely restored in twenty-four hours. (E. Gatti in "*Gaz. degli Ospital*," 1880–1, p. 4).

CAFFEIN.—Hutchinson (in the "*Centralblatt für Augenheilkunde*," Aug., 1887, p. 240) claims to have seen amblyopia closely resembling quinine amblyopia resulting from caffein.

ERGOTINE also produces ischaemia of the retina with consequent impairment of vision, by reason of its well-known effect upon the blood vessels. The possibility of such a result should be remembered when prescribing this drug for the cure of uterine neyomata, etc., especially when a disposition to such a condition already exists.

Among other drugs which produce more or less impairment of vision may be mentioned the following:

Osmic Acid causes great and sudden but temporary amblyopia, with loss of the faculty of accommodation, and with no ophthalmoscopic changes in the retina or optic nerve.

Antipyrin.—Blindness lasting one minute was reported after a dose of 15 grains. De Schweinitz reports the following visual disturbances noted by himself after taking large doses of the drug, viz.: "undulations in the atmosphere, something like those caused by the ascent of heated air, followed by an apparent shower of sparkling points of light; phenomena, in other words, which are the frequent prodromes of migraine."

Bromide of Potash has produced sudden loss of sight with pallor of the optic disc and narrow vessels.

Cannabis Indica, or hashish, frequently causes toxic amblyopia with symptoms resembling those occasioned by tobacco and alcohol. Unlike those substances, its effects are often unilateral, and there may be simply a scotoma without color disturbance. The visual symptoms may be associated with mydriasis or myosis, and disturbance of the accommodation.

Carbolic Acid produced complete blindness lasting two days in a man, after washing out the pleural sac with 100 grammes of a 3 per cent. solution (see "*Berlin-Klin. Wochenschr.*," 1882, XIX, p. 748). It causes also sluggish pupils which may be either dilated or contracted, and without ophthalmoscopic changes.

COCAINE has produced amblyopia. Glaucoma has resulted from its protracted application to the nasal mucous membrane. Hence it should be remembered that such use of the drug is not entirely unattended with danger.

CREOLIN is said to occasion sometimes more or less impairment of vision.

CHLORAL HYDRATE has caused temporary amaurosis. Other visual disturbances similar to those observed in hysteria, dependent presumably upon paresis of the accommodation, are attributed to its influence.

DIGITALIS produces sometimes more or less cloudiness of vision, due principally to the mydriasis which it occasions. This knowledge may enable one to differentiate the drug influence from a direct sequence of a cardiac lesion.

ESERINE. Complete blindness, of short duration, has been observed after its instillation into the conjunctival sac.

FELIX MAS. When administering this drug as a taenicide, it should be borne in mind that several cases of blindness have been attributed to it. It produces complete loss of vision of both eyes, coming on within twenty-four hours, or after several days. At first there are no ophthalmoscopic changes, but subsequently atrophy of the optic nerves develops. This circumstance demonstrates that the visual disturbance is not a result of an influence exerted upon the visual

centres, but an affection of the optic nerve stem, a form of retro-bulbar neuritis. There are no characteristic symptoms which admit of a diagnosis of the cause of blindness, without the previous history and attendant symptoms.

MORPHINE. Complete blindness of both eyes was observed in a patient who had taken two grammes of morphine subcutaneously within five days. The papillae were cloudy and the arteries narrow.

RESORCIN causes disturbance of vision in poisonous doses.

VISUAL HALLUCINATIONS result from the influence of BELLADONNA and ATROPIN, especially in the dark. They are also very prominent in the intoxication from CANNABIS INDICA. COCAINE, AESCULUS, SANTONINE and STRAMONIUM also occasion them. With AESCULUS the visions are distressing, and with STRAMONIUM they frequently take the shape of bugs, snakes, etc., resembling the hallucinations of *mania a potu.*

COLORED VISION has been noticed after various remedies, especially the following:

VIOLET VISION is sometimes caused by CANNABIS INDICA.

YELLOW VISION is characteristic of SANTONINE. This results within ten or fifteen minutes after the administration of the drug, and it may be associated with mydriasis and inequality of the pupils. The yellow vision is preceded transiently by violet vision. All

shadows appear in the complementary color, which fact demonstrates that the peculiar visual disturbance is caused by a peripheral irritation, and that the central visual sense is intact.

YELLOW VISION also results from CHROMIC ACID, DIGITALIS, NITRITE OF AMYL and PICRIC ACID. With the last named drug this phenomenon has been noticed after a small dose taken internally, too small to produce coloration of the media, and lasting an hour. This circumstance would indicate that the xanthopsia was caused by an action upon the visual centre. It was not followed by blue or violet vision.

IODOFORM occasionally causes RED or BLUE VISION, and COLORED VISION has been caused by COCAINE.

COLOR BLINDNESS, of a greater or less degree, may be caused by QUININE and those other drugs which have been mentioned as producing visual disorders similar to quinine amblyopia, viz.: SALICYLATE OF SODA, ERGOT and CAFFEIN.

PHOSPHORUS. SPARKS and FLASHES of light result characteristically from this drug, and from BELLADONNA and SANTONINE.

MICROPSIA and DIPLOPIA have also resulted from the use of COCAINE.

HEMIANOPSIA has been recorded as a symptom following the inhalation of dilute HYDROCYANIC ACID, and after poisoning with CARBONIC OXIDE. In the lat-

ter case the loss of vision was in the lower part of the visual fields.

B. PUPILLARY PHENOMENA, DISTURBANCES OF ACCOMMODATION AND OTHER OCULAR SYMPTOMS CAUSED BY THERAPEUTIC AGENTS.

Mydriasis is caused by Belladonna and its alkaloid Atropin, by Daturine, Duboisine, Ergotine, Homatropine, Cocaine, Aconite, Digitalis, Hydrocyanic Acid and Cyanide of Potash, Hyoscyamus and its alkaloids Hyoscyamine and Hyoscine, by Stramonium, Gelsemium and Conium. Calabar bean in poisonous doses sometimes causes mydriasis, although marked myosis is its usual effect. This circumstance might possibly prevent a mistake in diagnosis. Quinine produces mydriasis preceded by transient myosis. Aesculus Hippocastinum, much used in the form of a cerate as an application to hemorrhoids, produces great dilatation of the pupils in connection with the distressing visions already alluded to. This may aid in the diagnosis of poisoning from eating horse-chestnuts, which sometimes occurs in children. Although myosis is considered pathognomonic of Opium or Morphine narcosis, the opposite effect, viz.: mydriasis, is observed quite frequently in those habituated to its use. It should be mentioned, however, that myosis is never absent in acute opium poisoning, and rarely in the chronic form.

Myosis results as a prominent symptom from the internal administration of the following drugs, viz.: Chloral (perhaps preceded by transient mydriasis); Calabar bean and its alkaloid Eserine, Pilocarpine, Jaborandi, Morphine and Opium in its various forms and Salicylate of Soda. Quinine produces a transient myosis which is soon followed by mydriasis.

Spasm of accommodation is usually associated with contraction of the pupil, and paresis of accommodation with dilatation, hence it is unnecessary to repeat the drugs which cause these conditions. It is worthy of remark in this connection that in chronic morphine habitués the paradoxical condition of spasm of accommodation associated with mydriasis sometimes occurs.

Among other ocular conditions following the employment of various therapeutic substances may be mentioned the following:

Ptosis lasting two weeks has been produced by Sulfonal, which has become a favorite remedy for insomnia. A knowledge of this fact may prove valuable. Partial ptosis has followed the use of Iodoform.

Twitching of the Lids is caused by Calabar bean, Eserine and Jaborandi.

Swelling of the Lids occasionally occurs as an effect of Chloral.

Conjunctivitis sometimes follows the use of Atropin and Bromide of Potash. Pustules may appear on the conjunctiva during the employment of the

latter drug, even when there are none upon the skin. Conjunctivitis with irritation of the lids has also been reported as a result of CHLORAL.

CHRYSAROBIN OINTMENT, when applied to the skin, may produce conjunctivitis without secretion. When it enters the conjunctival sac, the inflammation which follows is attended with profuse secretion.

RESORCIN also causes conjunctivitis.

YELLOWNESS of the CONJUNCTIVA is sometimes caused by PICRIC ACID.

EPIPHORA results from the use of IODINE and IODIDE OF POTASH.

CORNEAL ANAESTHESIA has resulted from the use of SULFONAL, QUININE and APOMORPHIA. The latter produces also cloudiness of the cornea.

CORNEAL SOFTENING has been observed in OPIUM smokers, as a part of the general marasmus thus caused.

KERATITIS has been reported as an apparent result of large doses of BROMIDE OF POTASH.

CATARACT has been caused by ERGOTINE and PILOCARPINE. The rapid development of cataract noticed during the jaborandi or pilocarpine treatment, should teach caution in their employment with elderly persons or those with incipient cataract. Opacity of the crystalline lens has been produced in rabbits by the injection of NAPHTHALIN, and MENTHOL in lethal doses is said to have a similar effect.

GLAUCOMA sometimes develops after the use of mydriatics, especially ATROPIN, hence the tension should first be tested, and any tendency to a glaucomatous condition be carefully investigated before employing such agents. COCAINE applied persistently to the nasal mucous membrane has apparently precipitated an attack of glaucoma, and several cases following its instillation into the conjunctival sac have been reported.

CHAPTER XI.

OCULAR AFFECTIONS RESULTING FROM POISONOUS SUBSTANCES NOT MEDICINAL, ADMINISTERED ACCIDENTALLY OR BY DESIGN, OR CONNECTED WITH CERTAIN EMPLOYMENTS.

More or less impairment of vision results from the following substances:

ANILINE. "MISTY VISION" has been recorded as an effect of poisoning with this substance, together with mydriasis and pigmentation of the cornea and conjunctiva.

ARSENIC. Persons employed in the manufacture of wall paper, Paris green, cosmetics and pigments often suffer from arsenical poisoning. The RETRO-BULBAR NEURITIS which it apparently causes, and to which reference was made in discussing toxic amblyopia, is characterized by a distinct paracentral scotoma for red and green, and great diminution of central vision with preservation of the normal limits of the periphery of the visual field. The ophthalmoscope shows sector-shaped decoloration of the temporal portion of the optic nerve. Very few cases of the sort have been reported, and the fact that retro-bulbar neuritis does not seem to exist among the arsenic eaters of Styria leads one to

question whether arsenic was the sole cause of the visual disturbances attributed to it.

LEAD. Chronic lead poisoning gives rise to a variety of visual defects. Some are only indirectly caused by the poison, being a sequence of vascular changes in the cerebral vessels, such as sclerosis and peri-arteritis, of hemorrhages, spots of softening, etc., or of the kidney lesions which lead causes.

Other visual phenomena are the direct result of the poison upon the eye and the optic nerve, or upon the visual centre. The indirect influences alluded to will not be discussed here. The reader is referred to other portions of this work for their consideration.

As direct result of chronic lead poisoning we note :

1. Temporary dimness of vision, and also RETRO-BULBAR NEURITIS with its characteristic symptom of cerebral scotoma. Both the acute and chronic forms have been recorded as due to chronic lead poisoning. The amblyopia occurring with acute lead poisoning is usually of uraemic origin in consequence of a kidney lesion.
2. NEURITIS with a diffusely reddened and cloudy papilla, sometimes with hemorrhages and leading later to
3. ATROPHY of the optic nerve and complete blindness.
4. HEMIANOPSIA, CONCENTRIC NARROWING of the VISUAL FIELDS, COLOR DISORDERS, HALLUCINATIONS and

HYSTERICAL VISUAL DISTURBANCES occasionally develop.

Other symptoms of lead poisoning will usually be present to establish the diagnosis, such as colic, wrist-drop, headache, etc.

Saturnine poisoning may result from the ingestion of food or drink impregnated with lead, by handling paint, etc., by the use of hair dyes and cosmetics; and De Schweinitz says: "Not a few cases occur in tailors and seamstresses who bite instead of break the threads they are using. These threads are weighted with sugar of lead, which is thus gradually introduced into the system. I have seen one such case, in which, in addition to the general lead toxaemia, there was optic neuritis."

NITROBENZOL is used in the manufacture of dyes. It has an odor and taste very much like that of the oil of bitter almonds, and is sometimes used as a substitute for the latter in the manufacture of confectionery and in the preparation of perfumery. It produces DIMINUTION of VISION and CONCENTRIC NARROWING OF THE VISUAL FIELD resembling quinine amaurosis. A characteristic ophthalmoscopic appearance accompanying the amblyopia, described by Litten, is a darkening of the background of the eye "as if stained with ink" (see "*Centralbl. für prakt. Augenheilk,*" 1891, XV, p. 118). Sometimes it produces a central scotoma. Identical visual disturbances are caused by DI-NITRO-

BENZOL, which is used in the manufacture of explosives, especially "robarite" and "sicherheit."

BISULPHIDE OF CARBON AND SULPHUR CHLORIDE, employed in the manufacture of rubber, cause RETRO-BULBAR NEURITIS, as has been already mentioned. The former is the more poisonous. It is stated that 40 per cent. of the cases of chronic poisoning from this substance have amblyopia. The patient complains of an indistinctness of vision as if he were looking through a fog. This increases and a central scotoma with intact boundaries of the field develops, reminding one of tobacco amblyopia. The scotoma is most marked for red or green, and in mild cases there may be color blindness or contraction of the color fields without scotoma. MONOCULAR POLYOPIA, MACROPSIA, MICROPSIA, HEMERALOPIA, NYCTALOPIA and ANAESTHESIA of the CONJUNCTIVA and CORNEA have also been attributed to the influence of this poison.

Exposure to the vapor of dilute HYDROCYANIC ACID is reported to have caused temporary AMAUROSIS and HEMIANOPSIA.

SNAKE POISON causes an acute hemorrhagic diathesis, and either temporary or permanent IMPAIRMENT of VISION may result in consequence of hemorrhage in the retina or optic nerve, or in the visual centres. Amaurosis after snake bites is said to be of common occurrence in Brazil.

METHYL ALCOHOL, obtained by the destructive distillation of wood, a by-product in the manufacture of charcoal, and used as a solvent for resins and in the manufacture of aniline dyes, has caused BLINDNESS within twenty-four hours of its administration, according to Mengin. (*"Rec. d'Ophth.,"* 1879, p. 663).

MERCURY causes LOSS OF VISION due to hemorrhages, fatty degeneration and inflammation of the retina and optic nerve, resembling retinitis albuminurica.

PHOSPHORUS, used in the manufacture of matches, etc., causes very similar pathological conditions to those resulting from mercury, and with corresponding visual disorders.

LIDS. OEDEMA AND ECZEMA of the lids is caused by ARSENIC. The skin is often dry and scaly.

A BLUISH COLOR of the skin of the lids is caused by NITRATE OF SILVER.

CONJUNCTIVA. INFLAMMATION and ULCERATION of the conjunctiva is also caused by ARSENIC. It occasions a BROWNISH-RED COLORATION of that membrane, especially noticeable in workers in artificial flowers. The bluish hue noticed upon the skin of the lids in nitrate of silver poisoning, is also seen upon their inner surfaces. ANILINE causes a BROWNISH color of the conjunctiva and cornea, and a JAUNDICED appearance results from PHOSPHORUS.

PARALYSIS of one or more of the external eye muscles, of the iris and ciliary muscle, and of the lid may be an accompaniment of LEAD poisoning. The following case reported by Bach, of Wurzburg, in the "*Archiv für Augenhielkunde,*" April, 1893, is interesting and suggestive. The patient had exhibited symptoms of lead poisoning for some time, and the ocular condition was regarded as another manifestation of the same toxaemia. The pupils were irregular, and there was absence of the usual reflex contraction with convergence and accommodation and upon the stimulus of light. The reflex dilatation attending stimulation of the sympathetic was preserved. The visual field for white was normal, but was contracted for red and green. There was some protrusion of the right eye suggestive of Basedow's disease. There was paralysis, more or less complete, of the rectus internus, superior and inferior, and of the levator of the upper lid of the right eye, and slight paresis of the external rectus of the left eye. De Schweinitz asserts that the external rectus is more frequently affected than any other of the ocular muscles.

NYSTAGMUS sometimes accompanies poisoning by ARSENIC, LEAD and BENZINE.

PUPIL. CYANIDE OF POTASH (used in photography) and HYDROCYANIC ACID cause enormous DILATATION and immobility of the pupils, associated with slight exophthalmus, swelling of the upper lid, and a

peculiar staring expression. Mydriasis also results from ANILINE, BISULPHIDE OF CARBON, NITROBENZOL and DI-NITROBENZOL.

IRITIS has been caused by FUCHSIN and ANILINE.

VITREOUS OPACITIES occur in ARSENICAL POISONING, and according to Wolfe (see "British Medical Journal," 1879, II, p. 328) from excessive TEA drinking.

RETINAL HEMORRHAGES are caused by ANILINE, PHOSPHORUS, CARBONIC ACID GAS, LEAD, MERCURY, NITROBENZOL and SNAKE POISONING. With aniline and nitrobenzol the blood is very dark. With the former the fundus appears as if filled with ink.

CHAPTER XII.

OCULAR AFFECTIONS DUE TO POISONOUS SUBSTANCES CONTAINED IN CERTAIN ARTICLES OF FOOD AND DRINK. A: FUNGUS POISONING. B: PTOMAINE POISONING. OCULAR SYMPTOMS ATTENDING AND FOLLOWING ANAESTHESIA.

A. FUNGUS POISONING. Certain varieties of mushrooms are poisonous, and, in addition to the gastro-intestinal disturbances and other toxic manifestations, give rise to the following eye symptoms which may occasionally serve as an aid to an understanding of the systemic affection, and of the species of fungus which is the exciting cause.

PUPILLARY phenomena are among the most frequent of such symptoms.

MYDRIASIS follows the ingestion of some varieties of fungi belonging to the genus MORCHELLA, and it is usually accompanied with paresis of accommodation.

MYOSIS AND SPASM OF ACCOMMODATION attend poisoning with certain varieties of AGARICUS, a genus of mushroom embracing over a thousand species, many of which are edible. The poisonous varieties contain muscarine which occasions the ocular symptoms.

VISUAL HALLUCINATIONS are also symptomatic of fungus poisoning and AMBLYOPIA without organic changes in the fundus has been observed.

HEMORRHAGES AND FATTY DEGENERATION of the RETINA are occasional manifestations of the same toxaemia.

B. PTOMAINE POISONING. By this term is understood the effects produced by the ingestion of toxic substances developed in animal tissues and secretions during the process of decomposition. Ptomaines are produced through the agency of micro-organisms. They are the virus of the bacteria of putrefaction, and are found in the early stages of decomposition, sometimes before such a condition is perceptible to the senses. They develop notably in mussels, oysters, eels and other varieties of fish, also in sausage, ham, mutton, veal, beef, canned meats, and in milk and articles made from milk, as cheese, custard and ice-cream. However caused, the phenomena of ptomaine poisoning are very similar. Such poisoning is often serious and sometimes fatal. Typhoid fever is sometimes closely simulated.

The ocular symptoms of ptomaine poisoning are similar to those mentioned under fungus poisoning.

Congestion of the conjunctiva attends acute ptomaine poisoning.

Paralysis and paresis of the levator of the upper lid (ptosis), and of the external ocular muscles some-

times occur. Such symptoms are usually of nuclear origin and suggest hemorrhage or meningitis.

Mydriasis and paresis of accommodation usually attend ptomaine poisoning, and thus are of diagnostic importance. They are among the typical symptoms of poisoning by bad meat. They may be the only symptoms in mild cases, and are frequently overlooked. The mydriasis is always bilateral. Paresis of accommodation may occur without dilatation of the pupil.

Myosis and spasm of accommodation attend poisoning from neurine, a ptomaine found in putrefying fish.

Vision is not disturbed as a rule, although amblyopia has been noticed.

Some fishes are poisonous only at certain times and under certain conditions, when they may give rise to symptoms of ptomaine poisoning. The toxic element may reside only in special organs, as the liver and sexual glands, or may be dependent upon the kind of food which they have eaten. Decomposing haddock contains muscarine, and causes symptoms identical with those attending agaricus poisoning. De Schweinitz explains many of the symptoms of ptomaine poisoning by the statement that "many ptomaines are basic compounds, closely simulating the vegetable alkaloids, such as nicotine, atropin, veratrine and strychnine," and he adds, "consequently the ocular symptoms which may arise

under their influence are similar to those which the alkaloids themselves produce."

OCULAR CONDITIONS ATTENDING AND FOLLOWING ANAESTHESIA.

The behaviour of the pupil during anaesthesia from chloroform is important, and should be understood and carefully noticed, as it affords a valuable indication of approaching danger.

During the early stage, before narcosis is complete, the pupil is dilated and responsive to light. In the stage of complete insensibility, the pupils are contracted and the eyeballs are fixed. Dilatation with reaction to light returns with returning consciousness. Dilatation during narcoses indicates a necessity for caution.

Sudden dilatation during complete anaesthesia is an indication of impending asphyxia. The inhalations should be at once discontinued and every effort made to stimulate respiration and to avert imminent death. The concomitants of this form of dilatation serve to differentiate it from that previously mentioned, and to emphasize the danger attending it. They are: 1, profound narcosis in distinction from commencing narcosis or recovery from it; 2, absence of conjunctival and all other reflexes, instead of the presence of contraction upon the stimulus of light and of other reflexes; 3, stertorous respiration in

distinction from shallow respiration and efforts at vomiting; 4, fixed, immovable eyeballs, instead of mobile.

Arthur Ward, in the "London Lancet," for July 28, 1896, offers the following explanation of the pupillary phenomena: He considers the primary dilatation due to reflex inhibition of the unaffected third nerve centre induced by mental, sensory or sympathetic influences acting upon the semi-narcotized cerebrum. Dilatation he considers due to reflex inhibition. In complete narcosis, the cerebrum is no longer capable of receiving or transmitting peripheral impressions, but the third nerve centre still controls the pupil. In profound and dangerous narcosis, the third nerve centre is also narcotized and no longer controls the pupil which dilates and grows insensitive to light, and the globe becomes fixed. When the narcosis is not very profound, the contracted pupils will dilate after cutaneous irritation or when the patient is loudly called.

ETHER. Contraction is the rule during ether narcosis. In 1200 inhalations Jacob (*Jahrbuch für Augenheilkunde*, 1879, p. 229) observed mydriasis only six times.

ETHYL CHLORIDE has produced long-continued dense corneal opacities due solely to oedema.

ETHYL DICHLORIDE. Acute glaucoma has followed anaesthesia from this agent, associated with opacity of the cornea, but with slight external signs of inflammation.

Ethyl Nitrite produces dilatation and immobility of the pupils.

Nitrous Oxide. Extreme myosis attends the coma caused by inhalation of this gas. It also causes agreeable visual hallucinations. Dilatation of the retinal arteries and extreme redness of the papilla has been observed.

CHAPTER XIII.

BIBLIOGRAPHY.

The following is a partial list of authors and publications consulted in the preparation of this treatise.

"AMERICAN JOURNAL OF OPHTHALMOLOGY."

"ANNALES D'OCULISTIQUE."

ANTONELLI:

"Archives de Neurologia." Paris, Nov., 1893.

"ARCHIVES OF OPHTHALMOLOGY." (KNAPP & SCHWEIGGER).

ANDROGSKY, OF ST. PETERSBURG:

Zehender's "Klinische Monatsblätter für Augenheilkunde." Stuttgart, Vol. 32, p. 263, 1894.

ARNDT:

"System of Medicine."

BULL:

"Trans. American Oph. Soc." and N. Y. "Medical Journal," August, 1893.

BACH, LUDWIG:

Knapp's "Archives," January, 1895.

BERRY:

"Trans. Oph. Soc." United Kingdom, Vol. VII, p. 91.

"BERLINER KLIN WOCHENSCHR."

BACH, OF WURTZBURG:

"Archiv für Augenheilkunde," April, 1893.

BERGER, E.:
"La Médecine Moderne," Nov. 21, 1895.

BAKER, OF SAN DIEGO:
"Southern Cal. Prac.," January, 1893.

BAKER, OF UTICA:
"American Journal of Insanity," April, 1893.

BROWNE, LENNOX:
"Diseases of the Nose and Throat."

BURNETT:
"System of Diseases of the Eye, Nose and Throat."

CORTE:
"Deutsche Med. Woch.," January 23, 1891.

CLAIBORNE:
"Medical Record," August 14, 1894.

DE SCHWEINITZ:
"Toxic Amblyopias," 1896.
" "Transactions of the American Ophthalmological Society" for 1891, p. 23.

DOWLING:
"Quarterly Journal of Inebriety" for January, 1893.

"EYE, EAR AND THROAT JOURNAL."

ELSELING, OF GRATZ:
"Fortschritt. der Medicin." Berlin, Dec., 1892.

FUCHS:
"Text-book of Ophthalmology."

FOX, WEBSTER:
"Medical Bulletin."

FOSTER:

"Text-book of Physiology."

GOWERS:

"Diseases of the Nervous System."

GALEZOWSKI:

"Jahrb. f. Aug.," 1883, p. 297.

On Hereditary Ocular Syphilis. "Transactions of Dermatology and Syphilography." Paris, Nov. 15, 1895.

GRUBER:

"Diseases of the Ear." Translation. 2nd American Edition.

GATTI, E.:

"Gaz. degli Ospital," 1880, I, p. 4.

GRAY:

"Anatomy."

GOODNO:

"Practice of Medicine."

HOWE:

"American Journal Ophthal.," Vol. II, 5–6, 1885.

HANGG:

"Dis." Strasbourg, 1890.

HENSCHEN:

"Klinische und Anatomische Beiträge zur Pathologie des Gehirns." Upsala, 1892.

HUTCHINSON:

"Centralblatt für Augenheilkunde," August, 1887, p. 240.

HULANICKI, OF ST. PETERSBURG:
"Medicinische Wochenschrift.," June, 1893.

HERREN, OF JACKSON:
"Ophthalmic Record," January, 1893.

HAMMOND:
"Diseases of the Nervous System."

"JOHN HOPKINS HOSPITAL BULLETIN," Baltimore.

JACOB:
"Jahrbuch für Augenheilkunde," 1879, p. 229.

JENNINGS:
"Color Vision and Color Blindness."

"JOURNAL OF OPHTHALMOLOGY, OTOLOGY AND LARYNGOLOGY."

JULER:
"Ophthalmic Science and Therapeutics."

KNIES:
"The Eye in General Diseases."

LA GRANGE:
"Arch. d'Ophthalm.," January, 1887.
" "Medical and Surgical So.," Bordeaux, November, 1894.

LETTIN:
"Centralbl. for prakt. Augenheilk.," 1891, XII, p. 118.

LEWIS, E. PARK:
"Eye, Ear and Throat Journal," January, 1895.

LOPEZ:
"Archiv. f. Aug.," XXII, 2 and 3.

LORING:
"Text-book of Ophthalmoscopy."

MARPLE:

"New York Medical Record," March 11, 1893.

MILLS, CHAS. K.:

"International Clinics," October, 1895.

MOTT:

"International Clinics," 1895, Vol. I, p. 127.

MENGIN:

"Rec. d'Ophth.," 1879, p. 663.

MILES:

"Weekly Medical Review," 1884.

MADDOX:

"Ophthalmological use of Prisms."

MUSEHOLD:

"Deutsche Medicinische Wochenschrift.," February, 1892.

MAUTHNER:

"Sympathetic Diseases of the Eye."

" "Gehrin und Auge."

"NEW YORK MEDICAL JOURNAL," 1893.

NOYES:

Editorial note in Knies' "The Eye in General Diseases."

" "Diseases of the Eye," p. 683.

NORRIS AND OLIVER:

"Text-book of Ophthalmology."

NORTON:

"Ophthalmic Diseases and Therapeutics."

OLIVER:

"Medical News," November 11, 1893.

D'OENCH:

"N. Y. Medical Record," July 14, 1894.

" "Ophthalmic Review."

OSLER:

"Practice of Medicine."

OLIVER, CHAS. A.

"Ophthalmic Methods for the recognition of Nerve Disease."

PISCHL.

"Medical News," July 20, 1893.

RANDOLPH:

"Johns Hopkins Hospital Bulletin," June and July, 1893.

RANNEY, OF NEW YORK:

"Medical Record," May 12, 1894.

ROOSA, ST. JOHN:

"Treatise on the Diseases of the Eye."

SABRAZES:

"Semaine Médic.," September 26, 1894.

SEMELING:

"Charité Annal.," XI, p. 389.

STEWART, OF CINCINNATI:

"Eye, Ear and Throat Journal."

SOUS:

"Journal de Médecine de Bordeaux," Nov., 1893.

SILEX:

At Medical Society of Berlin, January 23, 1895, reported in "Annales d'Oculistique."

UHTOFF:

Graefe's "Archives," Bd. XXXII, *et. al.*, (p. 3, p. 6b).

VIALET:

"Annales d'Oculistique." Paris, April, 1894.

VERRAY:

"Rev. Med. de la Suisse Romande."

WOLFE:

"British Medical Journal," 1879, II, p. 328.

WARD, ARTHUR:

"London Lancet," July 28, 1896.

WHITE:

"Medical News," July 15, 1893.

WOOD:

"N. Y. Medical Journal," July 7 and 14, 1894.

WHITE, OF RICHMOND:

"Burnett's Treatise on Diseases of Ear, Nose and Throat."

WHITE, JOSEPH:

"London Medical Press and Circular," March, 1894.

ZIMMERMANN, OF MILWAUKEE:

Knapp's "Archives," January, 1895.

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